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COMMON OCULAR CONDITIONS IN GENERAL PRACTICE.[1]

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Perth.

IN this paper I have endeavoured to give you a brief review of symptoms, both general and local, of which a patient may come to his family doctor complaining and which are usually found to be primarily traceable to some ocular condition; and in some cases I have indicated, when it seems advisable, a rough differential diagnosis, especially when the treatment in the different disorders varies widely.

In dealing with matters appertaining unto a specialized subject, such as ophthalmology, one has to watch one's step somewhat carefully or, transferring the metaphor to the sea, steer a ticklish course between the Scylla of uninspiring platitudes and the, to my mind, even more perilous Charybdis of unintelligible technicalities; and if in avoiding Charybdis I have hugged Scylla somewhat closely, I must be pardoned. The question is whether the audience is to be lulled to sleep with oft-told tales or ruthlessly choked to death with indigestible masses of technical facts. Were I one of the victims, I should choose the former, and, gentlemen, if you tonight feel a pleasant drowsiness stealing over you, you may give way to it without the slightest qualm, since I willingly accept responsibility in advance and pronounce full absolution.

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on May 20, 1931.

Refraction.

I think it is probably better if we commence with the "common or garden" conditions and work through to the more rare. As an example of the former class we cannot do better than begin with the protean manifestations of errors of refraction, both spherical and cylindrical. Either alone, or more commonly both combined, can elicit these manifestations. The former (spherical) are exemplified in cases of hypermetropia and myopia. Hypermetropia is a condition in which the refractive power of the eye is deficient, rays are brought to a focus behind the retina and distant objects are seen better than near. Myopia is the exact opposite of all these. The latter is exemplified in cases of astigmatism, the condition in which rays cannot be focused to a point. There is in this condition a difference of curvature in successive meridians of the refracting surfaces of the eye, usually the cornea, the meridians of greatest and least curvature being usually, though not invariably, the vertical-horizontal. Whether they are vertical, horizontal or inclined at other angles, in the vast majority of cases the meridians of greatest difference are at right angles to one another. In the fortunately rare instances when this is not so, and which are usually secondary to injury to the cornea, the error is not accurately remediable by glasses. Various combinations of these differing meridia may be met with: (i) one meridian emmetropic, the other hypermetropic, that is, simple hypermetropic; (ii) one may be hypermetropic, the other myopic; mixed astigmatism; and so on through the other variations.

All varieties of astigmatism have this in common, that very low degrees can produce the most disproportionately distressing symptoms; this is not so much noticed in the lower degrees of uncomplicated spherical error.

The signs and symptoms of refractive errors, which for brevity's sake we will consider together, are so manifold, that it is difficult to know where to start. Eye strain from refractive errors is simply a form of peripheral irritation and, like other forms, it has reflex consequences which vary enormously with the individual and with his current state of health. Perhaps headache, usually most marked towards the end of the day, and in mild cases passing off after a night's rest, is the commonest and most typical complaint. We have all seen and listened to the sad story of the harassed and run-down mother of a large family, now getting on in her forties, who says: "I like to do a little reading or sewing in the evenings after work, doctor, but I don't seem to be able to keep my eyes open, and I get such terrible headaches." Asked where, she will generally brush her hand across her forehead or occasionally point to one or both eyes and say: "Right in there." She is probably a simple presbyope, the presbyopia being complicated or not with astigmatism, and the effect of loss of elasticity of the lens, through sclerosis, which begins in all subjects about forty, is complicated in her case by fatigue of the atonic ciliary muscle. Then at the other end of the scale is the young man or woman just beginning a business career of typewriting or clerical work, who complains of tiredness of the eyes and headaches, most marked in the evening, which completely disappear after a night's rest. These are usually astigmats, and the longer hours and sedentary habits of a city life bring to light refractive errors which almost certainly existed during school days, when, however, the shorter hours and healthier conditions enabled the eyes to cope with the disability without causing marked symptoms.

A point which has aroused my interest is the fact that moving pictures at an ordinary cinematograph entertainment, which are viewed as a rule from a considerable distance, and therefore call for no appreciable accommodative effort, appear to bring to light in a most distressing way even very small cylindrical (that is, astigmatic) errors of refraction. Objects seen at the cinema are as a rule not much smaller than life size at the distance, and discrimination of fine points of detail is not often called for, yet patients who would not have to wear spectacles for ordinary distance work find them essential at the cinema. True, movement takes place in two planes of space instead of three, but I hardly think that would account for it. Remembering, however, that movement on the screen takes place in a series of jerks, although these are with the present machines imperceptible, can it be that these rapid jerks subconsciously enhance the effect of the small errors? This suggestion was somewhat roughly dealt with by a neurological colleague to whom I offered it, and he thought that the frequent and sudden complete change of scene, for example, from caption to picture and *vice versa*, apart from the infinitesimal change from individual photograph to photograph, was more likely to be the cause. Perhaps members of the audience have their views.

Other more indefinite, but none the less distressing sequelæ of refractive errors are best summed up on the one easily comprehensible term, "indigestion," including gastric discomfort and loss of appetite; and as a consequence of these the whole mental and bodily tone is lowered; this reflects itself in the atony of the ciliary, amongst other muscles, rendering it less capable of coping with the unfair amount of work already imposed upon it, and thus completes the vicious circle. Bearing these arguments in mind, it is not difficult to credit the sometimes astounding tales of improvement in general health which some patients will relate after having a troublesome and long-standing error corrected.

Local signs are by no means wanting. A patient with chronic blepharitis, with or without styes, which does not yield to ordinary medical treatment and for which no occupational cause can be found, should always have the refraction done, especially children, in whom subjective investigation is not always satisfactory. Conjunctivitis (red eye) and epiphora (watering) generally go hand in hand with the above condition as signs of refractive

trouble, but may exist separately. The secretion is usually watery and seldom mucopurulent, except in the presence of a coexisting specific infective agent.

Another distressing condition which perhaps is more likely to come to the notice of the oculist during the investigation of a patient showing some or all of the symptoms detailed above, is that known as heterophoria or, in plain words, lack of muscle balance in the extrinsic ocular muscles, the recti and obliques. The maintenance of correct binocular vision in all positions of the eyes is an exceedingly delicate business, and if from some congenital or acquired reason an individual or group of these muscles is weaker or stronger than its opponents, the patient will have to maintain a permanent and conscious effort to counteract this, sometimes in the end unsuccessfully, with the result that binocular vision fails and squint and diplopia will appear. Even when the attempt is successful, asthenopia and eye strain occur, and sooner or later the patient must seek relief. This heterophoria is of several varieties, according to the plane in which the latent deviation lies; perhaps the commonest met with is diminished power of convergence. Briefly, the condition is investigated by placing a prism in front of one eye, causing artificial diplopia and loss of the stimulus to binocular vision, with the result that the eyes take up the position of rest and the latent deviation becomes manifest.

Glaucoma.

Another exceedingly important and dangerous condition of the eyes which shows in some cases marked general symptoms, is glaucoma, particularly the acute variety. This condition, it is scarcely necessary to mention, is characterized by increased intraocular tension, the cause of which is not always discernible, these cases being generally grouped under the heading of primary glaucoma, the cases which are secondary to some demonstrable intraocular pathological condition being termed secondary. In even the acutest attack, as a rule, premonitory symptoms can be elicited, the commonest and most striking being the presence of coloured rings round lights, this being naturally observed most at night. It is a refractive phenomenon, and caused in this case by disarrangement of the normal relation of the corneal lamellæ as a result of the increased pressure. It is not pathognomonic of glaucoma, and may occur in conditions where flakes of mucus overlie the cornea, and also in certain conditions of the surrounding atmosphere, for example, excessive dust. I can remember getting a great scare some years ago when my knowledge of ophthalmology was less even than it is now.

It was in the Sudan, where dust is excessive, and we had just halted a caravan of cars after a long ride in the desert and my eyes were feeling the strain of night driving. I happened to look at the head lights of a distant car and was horrified to see very beautiful and well marked haloes. It was, of course, the irregular refraction of rays due to the dust-loaded atmosphere, but in conjunction with the pain in my eyes it was enough for me, and I spent the next half-hour having my intraocular tension estimated by palpation until my bored colleagues went on strike. Needless to say, when the dust subsided, so did my coloured haloes.

In connexion with premonitory attacks, a history of previous transient periods of obscuration of vision is significant. The other signs and symptoms, such as cupping of the optic disc, arterial pulsation at the edge of the disc, perimetric changes, and microscopical corneal alterations are rather matters for special training and technique.

In the very acute attack the pain is so severe that it may cause vomiting, prostration and even rise of temperature; and unless care is exercised, the fundamental ocular origin of the disease may be missed and, a diagnosis having been made of bilious attack, colic or such like, time may be wasted in useless treatment, which, if rightly directed towards the eye condition, might have made the difference between total blindness and preservation of at least reasonable vision. In an attack as typical as the above, once the primary condition has been located in the eye, the diagnosis is fairly certain; an acute attack of iritis, which might have many signs in common with it, would not have, however, such a precipitate onset.

With glaucoma of more deliberate onset, on seeing the patient for the first time, perhaps so dazed by pain as to be incapable of giving a reliable history, the diagnosis in some cases may be difficult indeed, and particularly when an antecedent iritis has brought on a secondary glaucoma. The differential diagnosis is made all the more important in that the immediate treatment in the two conditions is diametrically opposite. Dilatation of the pupil, highly necessary in iritis, is disastrous in glaucoma, as it still further impedes the draining of the intraocular fluid and tends to intensify the hypertension already present. The condition of the pupil usually affords valuable diagnostic signs—in iritis usually small and irregular, in glaucoma large, immobile and vertically oval. Also, ocular tension is not as a rule raised in iritis, unless there be much accompanying cyclitis; in glaucoma it is always raised, and usually very markedly. The depth of the anterior chamber, that is, the distance between the back of the cornea and anterior surface of the iris, would, under the pathological conditions, be probably difficult to estimate, but, speaking broadly, marked shallowness would be suggestive of glaucoma and normal or unusual depth would be against it. Again, vision would be more affected in a case of glaucoma than in a case of iritis of comparable severity. Iritis may well occur at any age; glaucoma, at any rate the acute primary variety, is essentially a disease of late middle life.

Perhaps the most obvious and useful diagnostic sign of an uncomplicated iritis is the so-called circumcorneal ciliary injection, taking the form of a narrow zone of a dusky blue coloration immediately surrounding the cornea. The colour contrasts with the red of the conjunctival vessels, and the ciliary vessels, being deeper placed, do not move when the

conjunctiva is moved; after pressure the ciliary vessels fill quickly owing to their wide anastomosis, while the conjunctival, for an opposite reason, refill slowly. Unfortunately, in cases of acute glaucoma or very severe iritis there is so much accompanying conjunctival injection and œdema (chemosis) that this valuable diagnostic sign may be impossible to elicit.

Once acute glaucoma has been diagnosed (and in any doubt it is vastly preferable to treat the condition as glaucoma) no time should be lost in getting the patient under expert care, and preferably straight to hospital. In the period of waiting, isolation from noise and light, the recumbent position and morphine are indicated. If available, a drop of 0·5% to 1·0% eserine should be instilled into the normal eye in unilateral cases; if the condition is iritis, it will do no harm; in the case of primary glaucoma an attack in one eye will often, as a result of the pain and worry, precipitate an attack in the other eye, and eserine, previously applied to this eye, may well abort the attack. This is most important, and eserine promptly applied in this way has undoubtedly saved many an eye. The subsequent treatment depends on the course of the disease, but operative measures then or during a quiescent interval are essential in the vast majority. Stress may be laid here on the importance of mental worry and undue physical exertion in the ætiology of primary glaucoma; in a great many instances such factors have undoubtedly precipitated an attack in those predisposed. Heredity is also probably concerned, although authorities differ as to the relative importance placed on it, and, as a further point of interest, eyes predisposed to glaucoma are invariably noticeably small eyes. It must be understood that to buphthalmos or infantile glaucoma, a fortunately rare disease about the ætiology of which practically nothing is known, the above observations do not apply.

Cataract.

The next condition to which I propose to refer briefly is cataract, which is a disease characterized by total or partial opacification of the lens. There are many varieties, the commonest being senile, traumatic and infantile, in their order of frequency. The diagnosis is usually easy, particularly when central, the white opacity in the centre of the pupil being characteristic and the fundus reflex absent or poor. In uncomplicated cases the opacity is behind the plane of the iris, which rides freely over it, although the direct reflex is usually sluggish for obvious reasons. Senile cataract is uncommon before fifty, and is usually unilateral to commence with or more marked in one eye. Traumatic cataract occurs at any age, may be total or partial, and a history of a blow can usually be elicited, or a scar on the cornea or sclera may be seen. Infantile cataract may be of the lamellar type, usually bilateral, and considered to be due to intrauterine or post-natal malnutrition, and coming on in the first few years of life; or anterior polar, due to perforation of the cornea from intrauterine or post-natal gonorrhœal infection. Generally in these cases the corneal scar of perforation is obvious. Treatment, if indicated, is in all cases operative; no known medical measures will favourably influence any type of cataract, and this holds good even of the diabetic variety.

Conjunctivitis.

Conjunctivitis is a condition with which one is constantly called upon to deal. It is marked by redness of the bulbar and palpebral conjunctiva, varying in degree with the severity of the condition.

The different varieties are difficult to classify, but are generally divided into acute, including (*a*) simple, caused by irritants, dust, smoke, error of refraction *et cetera*; (*b*) mucopurulent (pneumococcal or staphylococcal infection); (*c*) purulent (gonococcal or pneumococcal); (*d*) membranous (gonococcal, streptococcal or diphtheritic); (*e*) phlyctenular; and chronic, including chronic simple, and other rarer conditions, such as tuberculosis, spring catarrh, syphilis, trachoma *et cetera*. The diagnosis of trachoma is important on account of its insidious progress and its highly infectious secretion. Its diagnosis, except in marked cases, is not simple; as valuable diagnostic signs may be mentioned: (*a*) a peculiar drooping of the upper lid, owing to its excessive weight, giving the patient a very characteristic sleepy appearance; (*b*) either extensive scarring of the conjunctival surface of the lids, particularly the upper, and more marked in the region of the *sulcus subtarsalis*; or (*c*) a very striking thickening of the palpebral conjunctiva of an intense dark red colour, so that this membrane strongly resembles the pile of a thick carpet. This condition may or may not be accompanied by the presence of the typical "sago grain" follicles in the upper fornix. With these phenomena there is often in long-standing cases added the condition known as pannus, that is, a superficial vascular membrane growing into the cornea from the periphery. In all types of conjunctivitis care must be exercised to detect the bluish circumcorneal flush indicating possible coincident inflammation of the uveal tract. A certain amount of conjunctivitis with epiphora is a frequent accompaniment of iritis or keratitis.

The treatment of conjunctivitis, like other diseases, varies with the specific cause, which must first be determined. In acute simple conjunctivitis the irritating agent, be it dust, foreign body, smoke *et cetera*, will be removed and a bland lotion (acid boracic 5% solution) or magnesium sulphate prescribed for a few days. If there is much secretion a bandage is contraindicated, as the secretion is thus dammed back and infection facilitated. In the specific infectious varieties, bacteriological examination is sometimes necessary when diagnostic clinical signs are absent or obscure. An acute mucopurulent conjunctivitis may well be staphylococcal, pneumococcal or due to the Koch-Weeks bacillus. Pneumococcal conjunctivitis is rather more commonly unilateral than the others, and may show

small petechial hæmorrhages on the bulbar conjunctiva. A further exceedingly important fact is that pneumococcal toxin seems to be the only one which can penetrate the intact cornea and cause an iritis. Colloidal silver salts (argyrol, protargol) are advised by some in this connexion; others condemn them as routine applications owing to the danger of argyrosis (an impregnation of the conjunctival tissues with metallic silver, which is permanent and most unsightly), and pin their faith to "Ethylhydrocuprein" (optochin) or irrigation with one in 10,000 perchloride of mercury solution. For staphylococcal and Koch-Weeks conjunctivitis respectively, frequent washing out of the conjunctival sac with oxycyanide of mercury solution one in 6,000, or perchloride of mercury one in 8,000, will generally effect a cure. In the severe purulent forms, gonococcal and streptococcal, frequent irrigations, judicious and careful paintings with silver nitrate, hot or cold applications, according to the clinical indications, are necessary, but these cases are always worrying, because of the imminent danger to the cornea, that is, possible ulceration followed by perforation. An exceedingly important measure in unilateral cases is the careful protection of the good eye by a shield. Membranous conjunctivitis is usually diphtheritic, when serum, locally as well as generally administered, is essential. These more severe affections, however, are much better referred to a specialist, owing to the grave and imminent danger to the eye.

The treatment of phlyctenular conjunctivitis, a common disease of ill-nourished children, is still a matter under discussion. Frequent irrigations with perchloride of mercury, the use of yellow oxide of mercury ointment and either tuberculin injections or staphylococcus vaccines have given good results, combined, of course, with energetic constitutional measures. Of the chronic varieties, chronic simple conjunctivitis is treated on general principles after removing the underlying cause. Errors of refraction must not be forgotten in this connexion. Of the others, in angular conjunctivitis, caused by the Morax-Axenfeldt bacillus, zinc salts are specific. Syphilitic and tuberculous conjunctivitis are not common, and treatment runs mainly on constitutional lines. That of trachoma is long and tedious, and consists essentially of applications of silver nitrate or occasionally in severe cases copper sulphate, and certain operative procedures for special complications.

In the treatment of conjunctivitis generally certain features stand out. As we have hinted, free secretion must not be dammed back, as this predisposes to further infection, and bandages must be replaced by dark glasses for protection until it has been controlled by astringents. Secondly, lotions in the treatment of conjunctivitis act in most cases mechanically by washing out infected *débris* from the sac, and not as antiseptics, for use as which they cannot be applied in sufficient strength; therefore, irrigations must be thorough, and introducing the lotion from an undine, the lids being held open with the other hand, is vastly preferable to trusting the patient to use the so-called eye bath.

Detachment of the Retina.

Detachment of the retina is a condition not difficult to diagnose without special instruments, and its early detection is important if treatment is to be successfully undertaken. A history of a blow or fall is usually given, and those with short-sighted or myopic eyes are more liable to this disability than others; indeed, in a high myope spontaneous detachments often appear to occur. The patient will say that a certain portion of his visual field is blank and he cannot see objects situated in it (negative scotoma) or that there is a black cloud of a certain shape in front of his eye (positive scotoma). Sometimes a detachment of the upper half of the retina will gradually change to one of the lower half, owing to the gravitation of the fluid between retina and chorioid, and the upper half of the retina becomes replaced, with restoration of function. On shining a plane mirror into the eye at a distance of about one foot, the normal red fundus reflex will be interrupted at the site of the detachment, and the latter, if well marked, will be seen to float forward like a grey carpet on which the vessels show clearly as black lines. It is a very striking appearance and, once seen, cannot be mistaken. It is most important to realize that a detachment may also be an indication of a malignant chorioidal or retinal neoplasm, and in this case early recognition of its nature is essential, as the patient's life probably depends on it.

Squint.

Squint or strabismus is defined as a condition of the eyes when the visual axes do not meet at the point of fixation, and there are two main varieties, concomitant, which concerns us here, and paralytic, which does not. In the vast majority of cases it is the former variety which is present when a mother brings a child to you with the information that he or she "has a cast in his eye, doctor." The information is usually superfluous and the deviation only too obvious, but some less marked cases take some care for their detection, and in yet others the squint becomes manifest only at certain times, for example, when the child gets tired. All these cases should be referred to a specialist at once, for refractive errors are commonly at the bottom of the trouble and delay may mean that the child loses permanently his faculty for binocular vision. In untreated cases the vision of the squinting eye may deteriorate markedly through disuse (*amblyopia ex anopsia*), although no organic damage can be detected in the eye. Paralytic cases are much more rare and much less tractable.

Foreign Bodies.

A communication of this nature would not be complete without some reference to foreign bodies in the eye, and by this one means in the conjunctival

sac as well as actually intraocular. Foreign bodies in the conjunctival sac, in which is included the surface of the cornea, are an everyday occurrence in all walks of life, and as a rule are removable by domestic methods. Some of these are less desirable than others, and amongst the former class may be included the procedure very prevalent in certain communities of removing foreign bodies with the tongue. This may be all very well in some cases, but in others, local chancres and other sequelæ equally undesirable have followed upon this repulsive practice. Of the said domestic methods, perhaps the corner of a clean handkerchief is as efficient as anything else for the removal of a foreign body loose in the conjunctival sac or resting on the cornea. If it shows the slightest disinclination to move from the latter situation, it should be left alone and the patient transferred to expert care. Of foreign bodies on the cornea 99% are very lightly adherent and the slightest touch with a spud will shift them; the hundredth is sticking into the substance of the cornea and may even be extending into the anterior chamber, from which, on its removal, all the aqueous humour will flow, with the most unpleasant consequences. When it is remembered that the cornea near its centre is something less than a millimetre thick, it will be understood that it is well nigh impossible, without special technique, to decide before removal whether a foreign body is entering into the anterior chamber or not. As a matter of fact, even normal corneæ may be only half that thickness, so it will be recognized that the margins of safety in the process of digging out foreign bodies are somewhat narrow. Accurate localization is possible only with the corneal microscope and the narrow beam of the slit lamp. With this technique in skilled hands a considerable degree of accuracy is obtained, of the order of fifths of a millimetre.

The symptoms of a foreign body in the eye are almost too well known to need recapitulation. The history, the watering of the eye, the reflex blepharospasm, the conjunctival injection, probably more marked in the region of the body itself, the pain and irritation are known to all of us probably by personal experience. The instinctive procedure is to rub the eye, Nature's idea presumably being to increase secretion by stimulation of the lachrymal gland and so wash out the intruder. Unfortunately, the usual result is to increase the damage done by the foreign body and cause the latter to become more firmly embedded. Such procedure should therefore be discouraged and immediate search made for the foreign body. The lower fornix should be searched first, partly because it is accessible, all that is necessary being to pull the lower lid gently outwards and get the patient to turn his eye up, and partly because the effect of gravitation makes it likely that a loose foreign body has lodged there. In this connexion we may mention that, although it seems almost incredible, it is quite a fairly common thing for a loose eyelash to become impacted in the lower punctum, that is, to become inserted like a probe, and unless looked for it may well be missed. Extraction is easy, as it never goes completely in.

Having "drawn blank" in the lower fornix, one looks under the upper lid, which can be everted by the expert with no inconvenience to the patient, and the *sulcus subtarsalis*, the site of election for the lodgment of foreign bodies, is investigated. This is a linear horizontal furrow on the conjunctival surface of the upper lid running parallel to and a few millimetres from the free edge. It is the frequency of lodgment of foreign bodies in this situation that explains the efficacy of the lay treatment of drawing the upper lid down over the lower and allowing it to fly back like a window blind, insuring at the same time that the conjunctival surface of the upper lid is in contact with the cutaneous surface of the lower. It is this friction which in the majority of cases draws the foreign body from its position.

If nothing is discoverable in this situation or in the region of the inner or outer canthus, the upper fornix must be investigated, and this is by no means as accessible as the lower, and consequently its examination is often omitted. Numerous cases of this have occurred, the irritation of the eye persists for months to the mystification of the medical attendant, and have even been known to terminate by the bursting of an abscess in the upper fornix and the extrusion of some gross foreign body into the conjunctival sac, such as an insect, husk of corn *et cetera*. The only ways of satisfactorily examining the upper fornix are: (*a*) by pulling the everted upper lid outward with some sort of blunt hook and shining artificial light into the cavity, or (*b*) by doubly everting the lid, that is, after the lid has been once everted, bending it back on itself once more. This is by no means easy, nor can it be considered pleasant for the patient. However, in any case of undiscovered suspected foreign body no pains must be spared to eliminate the upper fornix as a possible site.

Foreign bodies on the cornea have been alluded to and frequently need a magnifying glass or loupe for their detection. If they have made a considerable wound, they may be accompanied by hyperæmia of the iris and ciliary injection. Loss of corneal substance not reaching to Bowman's membrane may be regenerated without scar formation, although the regenerated surface may not be completely spherical in contour and a facet be left, which in certain lights may be clearly demonstrated. Injuries involving Bowman's membrane usually mean permanent scar formation, hence the importance of avoiding injury to it when fishing round for the removal of a foreign body.

Although the diagnosis and treatment of intraocular foreign bodies hardly come within the scope of general practice, a few remarks may be made in this connexion. If the foreign body is of any size the force necessary to penetrate the eye produces marked disintegration with, possibly, expulsion of the contents of the eye, including iris, lens

and vitreous. When the wound of entry is behind the visible portion of the globe and the ocular contents have herniated into the orbit, a sign suggestive of perforation is marked loss of ocular tension, palpable through the upper lid, and the sensation obtained by the finger is like that gained on pressing a child's rubber ball which has been punctured. Enucleation of such an eye is strongly indicated for a variety of reasons, but it is well to obtain expert advice to insure that the eye in question is really in a sufficiently desperate condition to justify such heroic measures. The common intraocular foreign bodies, iron, steel, copper and stone, are generally so small that special measures are necessary for their detection, and chief amongst these are the X rays, pictures being taken in two planes; by the interpretation of these by a method devised by an American, Sweet, a high degree of accuracy in localization is obtainable. Not infrequently, of course, they are visible by the ophthalmoscope, and this facilitates their recognition.

Any foreign body is a potential source of damage to the eye concerned, and unfortunately very frequently to the fellow eye as well, through the medium of that nightmare of the ophthalmologist, the much dreaded disease known as sympathetic ophthalmia. In brief, this essentially consists of a low grade uveitis with certain histological features of its own, starting in the injured eye and spreading by some means not yet understood to the other. The fundamental cause of sympathetic ophthalmia is still a matter under keen discussion; theories range from a staphylococcal septicæmia to an ultramicroscopic virus. It is more common in children. Perforation of the globe appears an essential factor and injury to the ciliary body seems to predispose strongly to the condition. Curiously enough, it is uncommon after panophthalmitis, that is, suppuration in the eye.

Prophylactic enucleation of an eye injured as above and in which vision is negligible is highly important. Timely enucleation of such an eye is an absolute guarantee against sympathetic disease in the other, though recently I saw a report of a case in *The Lancet* in which this rule did not hold good, whereas, once sympathetic ophthalmia has commenced in the good eye, enucleation of the injured one is frequently too late to be of any service.

The early diagnosis of sympathetic disease is one of the most difficult problems in ophthalmology, and here the slit lamp and corneal microscope are invaluable. By this means the very early dew-like precipitates on the back of the cornea and very slight haziness in the aqueous can be detected which are visible by no other means and whose timely detection may mean the saving of the sympathizing eye. My reasons for referring at such length to this condition are that many eyes are seen in general practice, particularly in industrial districts, in which perforation has taken place with or without retention of a foreign body. The eye may seem on a cursory inspection to have settled down nicely and to be giving no further trouble, while in reality the eye may be the seat of sympathetic trouble which may at any time flare up in the fellow eye with disastrous consequences. Such eyes should always be placed under expert care for either immediate enucleation or very careful watching. Particularly when the injured eye still retains fairly useful vision, the decision as to treatment is perhaps the most momentous one the oculist is called upon to make, and the earlier a patient is referred to him, the better equipped he is to deal with this very great responsibility. Once sympathetic disease has gained a hold upon an eye, enucleation of the exciting eye loses its value, and treatment of the sympathizer is exceedingly unsatisfactory; nothing but a miracle will prevent serious damage, and total blindness is only too common a result.

Apart from the effects of mechanical damage which have been referred to above, the consequences of the presence of certain foreign bodies in the interior of the eye vary in some cases with the nature of the foreign body itself. The best marked and most striking clinical entity is that known as siderosis due to the presence of a fragment of iron or steel in the eye. The pathological changes are slow and extend over months, and consist essentially of a permeation of the intraocular tissues with a solution of iron in the tissue fluids in the form of carbonate of iron. The metal is deposited mainly in the cornea, iris and retina. In a well marked case the change of colour in the iris is readily noticeable, changing as it does to a dull muddy brown which generally contrasts noticeably with the normal. In an enucleated eye treated by the ferro-ferricyanide test the blue colour is seen to extend through cornea, lens capsule, uveal tract and retina. The impregnation of the tissues by the metal is followed by degeneration and vision is soon lost. In this condition it is a useless and dangerous eye. Small metallic, that is, magnetizable fragments may often be removed by manipulation with the hand or giant magnet, according to their position. When this is performed early and completely, the prognosis after removal of a small body is good. The very high velocity necessary to enable a small fragment to pierce the outer coat of the eye generally sterilizes them by heat, so infective complications in these cases are not common. A piece of copper, although sterile, usually causes localized suppuration, leucocytosis being induced by chemical action. This process may, however, be long delayed, and the fragment may become encysted. Other substances, such as wood, stone, glass *et cetera* are not so commonly sterile, and an infective panophthalmitis is the sequel.

Epiphora.

Epiphora, or watering of one or both eyes, is a symptom which we have all seen and probably experienced at some time or other. The underlying possibilities of causation vary widely and are roughly divisible into two sets of conditions: (*a*) those causing increased secretion of the lachrymal

fluid with normal or interrupted outflow, and (*b*) those causing interruption of the outflow with normal or increased secretion. Of the first, a foreign body on the cornea or anywhere in the conjunctival sac is the commonest instance and is generally easily to be dealt with according to the directions given above. Of course, any form of irritation of the sac, including most of the various forms of conjunctivitis, will produce epiphora, but in a great many of these cases the increased secretion is not purely watery, but is mixed with other special pathological products, mucus, pus *et cetera*. Inflammation of the uveal tract, particularly the iris, is also associated with a reflex hypersecretion of tears, and, while on the subject of reflex lachrymation, mention must be made of emotional causes and painful peripheral stimuli, as of the nasal mucous membrane by pungent odours, or of the retina by very bright light. The second set of conditions mentioned was dependent upon some interruption of the drainage of tears from the conjunctival sac, and would include anything tending to narrow or occlude the lumen of the canaliculi or naso-lachrymal duct. Foreign bodies, hair or fungi in the canaliculi, acute or chronic inflammation with fibrosis, trauma, neoplasms, all play their part and generally show fairly obvious diagnostic signs.

An interesting and not particularly rare condition is seen in babies in whom a unilateral or bilateral epiphora, without inflammation or other signs, is early noticed. The eye is not red or painful, but just waters excessively. It is due to imperfect canalization of the lower end of the naso-lachrymal duct, which is developed from a solid cord of epithelial cells derived from infolded surface epithelium. The condition frequently settles of its own accord; failing this, one passage of a small probe usually effects a permanent cure. Under normal conditions, tears, as they are secreted, evaporate from the surface of the globe, and only when from emotional or reflex causes secretion is excessive, do tears pass down the naso-lachrymal duct. As a consequence, epiphora is not noticed under normal conditions after extirpation of the lachrymal sac, although sometimes it is hard to convince patients of this when seeking consent for an operation for dacryocystitis.

Another very common, very distressing and yet easily relievable condition causing epiphora is that known as ectropion, or the falling or drawing away of the lower lid from the globe. The commonest and most easily remedied is the senile form, in which originally the lower lid tends to fall away by gravity, aided by the lack of tone of the senile tissues. The conjunctival lid surface is thus exposed, chronic conjunctivitis supervenes with thickening of the lid and addition to its weight, more sagging outwards of the lid completing the vicious circle. Treatment consists of astringent applications to reduce the hyperæmia and conjunctivitis, and when this has been alleviated, an operation to restore the position of the lid, which as a rule gives good results.

Interstitial Keratitis.

Next we come to the consideration of a common and highly important condition which is responsible for a great deal of defective vision—I refer to the disease known as interstitial keratitis. To the average medical man it is associated with one ætiological factor and one only, the *Spirochæta pallida*, and while in most cases this will be found to be so, there are exceptions. After all, the name means no more than it implies, that is, a deep-seated inflammation of the corneal tissue, and pathological conditions identical with these associated with undoubted *lues* have been observed in late cases of tuberculous iritis and malaria. The clinical condition is seen occasionally in animals, arising apparently spontaneously. I recently saw what I took to be such a case in a dog belonging to a professional colleague. The condition was then unilateral, and no cause could be suggested by the owner.

It is essentially a permeation of the deeper layers of the corneal tissue with aggregations of lymphocytes and later by leashes of vessels arising from the ciliary system and arrayed in a somewhat characteristic manner.

Clinically, probably 90% or more of cases of interstitial keratitis are due to the spirochæte, which can sometimes be demonstrated in the corneal tissue. The vast majority of these cases are of the congenital variety, the subjects of it showing at the same time the well known stigmata, Hutchinson's notched incisor teeth (only permanent set), frontal bosses, deafness, tibial periostitis, saddle nose *et cetera*. The typical age of onset is from five to fifteen years, sometimes later, rarely earlier. The congenital variety is almost invariably eventually bilateral, although weeks or may be months may intervene. This is a practical point, for a definite warning to this effect, when a unilateral case appears, may prevent, when the condition becomes bilateral, complaints from irate parents that your treatment has caused the disease to spread to the other eye.

The treatment of the condition may be, indeed is, regrettably ineffective in arresting its course, but I do not think we can allow it to be stigmatized as definitely noxious! It is frequently, and some authorities consider invariably, associated with an accompanying uveitis, and these authorities are of opinion that the uveal condition is primary and the corneal secondary. When the disease is established, the cornea rapidly becomes steamy and opaque and has aptly been compared to ground glass. The uveal condition is thus masked. However, frequently, on examining the periphery of the fundus of the hitherto unaffected eye, spots of anterior chorioiditis may be seen, lending colour to the theory of the fundamental nature of the uveal lesion. The opacification of the cornea may commence anywhere, but in the course of weeks usually becomes complete, accompanied by ciliary hyperæmia and the growth of bright hued vessels from the periphery towards the centre, forming the

pink, so-called salmon patch which is very characteristic. Photophobia, lachrymation and probably conjunctivitis are usually superadded.

Treatment, as I have hinted before, is not encouraging. The usual constitutional antisyphilitic remedies appear quite powerless to abort the attack. Certain local measures, however, can be taken which limit the damage in the eyes. The course is chronic; after opacification has become complete clearing as a rule sets in and may progress slowly for eighteen months to two years. The clearing process seldom, however, goes very far, some degree of opacity persists, and after two years hope from further medical treatment may be abandoned. In certain selected cases a judicious iridectomy may result in improvement in acuity, but such cases are unfortunately rare. The deep vessels persist and may be seen, occluded as a rule, many years afterwards, and afford valuable confirmatory evidence of a previous luetic process. It is an interesting fact that trauma, not necessarily severe, is very frequently the exciting cause of an attack in those with the requisite diathesis, and some authorities regard the manifestation as evidence of an anaphylactic process.

Not so very long ago I had referred to me a very typical case of interstitial keratitis of undoubted congenital luetic origin. The patient had all the typical accompanying stigmata. The history was that the trouble had dated from the patient's getting an eyefull of gross foreign bodies while working with a chaffcutter. The interest of the case for me lay in the fact that substantial compensation had been paid him for his loss in visual acuity, which was fundamentally and entirely due to his inherited syphilis. Surely, I thought to myself, to paraphrase the Scriptures, a case of visiting the sins of the fathers upon the employers!

The cornea of such an eye is usually thickened in the acute stage and afterwards, in the period of recovery, it may become irregularly thickened or thinned. Such a cornea is unfitted to resist even normal intraocular pressure and may become ectatic, that is, exhibit localized bulgings.

The acquired syphilitic variety of this disease is considered a late secondary phenomenon; it is not common (5% of all cases of interstitial keratitis) and practically invariably unilateral. The symptoms and course differ in no important respect from the congenital kind, except that as the age incidence is higher, the clearing of the cornea does not tend to be so complete as in younger subjects.

Renal Retinitis.

Time now draws short, and I should have liked to treat of some retinal conditions. To consider many of them is obviously impossible, and after some deliberation I have decided to choose as typical a one as I can and one embodying in its manifestations as many examples of various retinal lesions as possible. I think renal retinitis meets the case. I prefer the term renal retinitis to albuminuric, as albuminuria may have sources of origin other than the kidney. A word of caution is here necessary. One has grown up to regard the suffix "itis" as connoting inflammatory changes in the tissue or organ concerned. In ophthalmology a good deal of laxity is shown in the use of this term, and retinitis and chorioiditis are used to describe pathological alterations in the retina or chorioid which may be the result of congestion or even merely degenerative changes. It is regrettable, but the procedure has become sanctioned by long usage, and I must ask my hearers to bear this in mind while listening to the descriptions which follow.

Some authorities, notably Batty Shaw, consider that (*a*) retinal arteriosclerosis, (*b*) the condition known as arteriosclerotic retinitis, and (*c*) renal retinitis show differences merely in degree, that all are due to a toxæmia acting with varying rapidity and in varying strength to produce these different lesions. What we are going to consider now are the retinal changes clinically associated with the various types of nephritis, leaving the physicians to dispute over the fundamental cause of the lesions. Let it be said at the outset that retinal changes are late manifestations of renal disease, and are therefore uncommonly, if ever, seen in primary acute nephritis. Their presence in any given case postulates that the condition is of fairly long standing. Foster Moore estimates that retinal changes occur in 33% of cases of sufficient standing and severity to warrant admission to hospital, and with increasing severity the percentage incidence of retinal changes also increases. These changes may also be regarded as of grave prognostic import. Foster Moore estimates that the great majority of patients showing them do not survive two years, and a great number of these less than one. They will be found almost invariably associated with high blood pressure and marked arterial changes. With the exception of the nephritis in pregnancy they are, as I have remarked, uncommon in acute nephritis and, of the chronic varieties, are much commoner in interstitial than in parenchymatous, though a variety of retinitis, with somewhat specially marked features, does occur in the latter, as will be described.

The actual retinal lesions may be described under the headings of œdema, hæmorrhages and exudates, and it is the varying preponderance of one or other of these three which marks the difference to the oculist between the various types of nephritis.

Let us consider first the manifestations of retinal œdema. It should be understood that normal retinal nerve tissue is transparent and is not visible by the ophthalmoscope; pathological or degenerative changes in it render it opaque, and this opacity shows as variously shaped whitish patches. The appearance of the patches differs when the process is a mere œdematous infiltration of the tissues and when there is definite hyaline degeneration. An example of the former condition is seen in the so-called cotton wool patches which are exceedingly suggestive of parenchymatous nephritis, although occurring sometimes in the other varieties. These patches generally surround the disc, are sometimes fringed with blood and sometimes are so marked as to suggest a heap of snow.

Broadly speaking, œdema, as represented by these cotton wool patches and by collections of subretinal

fluid causing flat detachment of the retina, is more typical of those varieties of nephritis in which the toxic factor is particularly marked, for example, parenchymatous nephritis and the nephritis of pregnancy. The whitish, hard-edged spots of exudate, on the other hand, either isolated or arranged in what we shall describe as the star figure, rather suggest the chronic interstitial variety. Foster Moore has made investigations into the comparative times taken by the various lesions to disappear from the fundus, and those deriving their origin from œdema fade much more readily than the hyaline patches of exudate. Involvement of the refractive media, for example, the vitreous, in the pathological process is not common, so the whole picture is as a rule clearly seen with the ophthalmoscope. In detachment of the retina due to œdema the fluid exuded from the vessels appears to collect in the subretinal space and float the retina forward. If this process happens to start in the upper portion of the fundus, it is common for it to gravitate to the lower half sooner or later. On the subsidence of the œdematous process, whether evidenced by detachment or cotton wool patches, restoration of function in the retina is common, and particularly good is the prognosis as regards vision in the eclamptic cases, provided requisite measures are taken early. So much for the retinal manifestations of œdema.

Exudates, as I have before mentioned, are whitish opacities in the retinal tissue, caused presumably by the localized action of the common toxin and taking the pathological form of hyaline degeneration. They are small, discrete, hard in outline, and lack the size and soapy, shiny appearance of diabetic spots. They may be scattered over the fundus, but the most typical arrangement is in the form of the so-called star figure. It consists essentially of a collection of variously sized and shaped spots of hyaline degeneration, and the reason of the peculiar arrangement has been the subject of much discussion. Some ascribe it to the anatomical disposition of the fibres in Henle's layer of the retina, but to my mind Foster Moore's theory of lines of tension from underlying œdematous fluid radiating from the macula, where the retina is supposed to be adherent to the chorioid, seems more probable. These radiating lines might then form a sort of foundation for the laying down of the spots of degeneration. This star figure, although very suggestive, is by no means pathognomonic of nephritis; it is found in some cases of cerebral tumour, particularly in children, and in thrombosis of the central artery and vein.

Apart from the signs of arteriosclerosis of the vessels, hæmorrhages are the last of the manifestations left to consider. In nephritis they are usually set in the superficial layers of the retina and therefore, owing to the anatomical arrangement of the nerve fibres, they are flame-shaped. They are most numerous and largest towards the centre of the fundus. Hæmorrhages in the deeper retinal layers, more typical of the diabetic state, also infrequently occur in nephritis and are circular in outline.

Well, we have considered one by one the nephritic retinal manifestations, let us combine them and note the result. It must not be thought, however, that we can combine the ingredients as in a prescription and say, here is a case of nephritis of such and such a class. Just as cases of nephritis themselves shade imperceptibly from one arbitrary classification into another, so do the retinal appearances show every possible permutation and combination of the above signs. Each case must be treated on its merits and the retinal appearances interpreted in the light of constitutional findings.

Papillœdema.

There is one manifestation I have missed, and that is papillœdema, an œdematous swelling of the disc with blurring of its edges. This may on occasions be unilateral. Diagnosis may sometimes be difficult—to cite an example, in a patient with unilateral star figure and papillœdema and accompanying headache and vomiting. Is the condition cerebral tumour or renal disease? In some instances the most careful investigation is necessary before the decision can be made. The progress of renal retinitis does not follow *pari passu* with the progress of the disease itself; subsidence of the retinal condition frequently occurs before the death of the patient and is of no prognostic import. As a point of interest we may notice that considering the severity of the lesions in the retina a surprising degree of visual acuity is often shown by these patients. It is also a fact, although somewhat difficult to explain, that renal retinitis, after subsidence, does not tend to recur, however long the patient may survive.

Conclusion.

That concludes my paper. It leaves me with the impression of sketchiness and disjointedness, but when time is necessarily limited, and many widely differing conditions must be treated of, cohesion is difficult to attain.

I am left hoping that you have had a tithe of the enjoyment in listening to this paper that I have had in preparing and reading it.

THE EFFECT OF COLLOIDAL LEAD ORTHOPHOSPHATE ON THE DOMESTIC HEN.[1]

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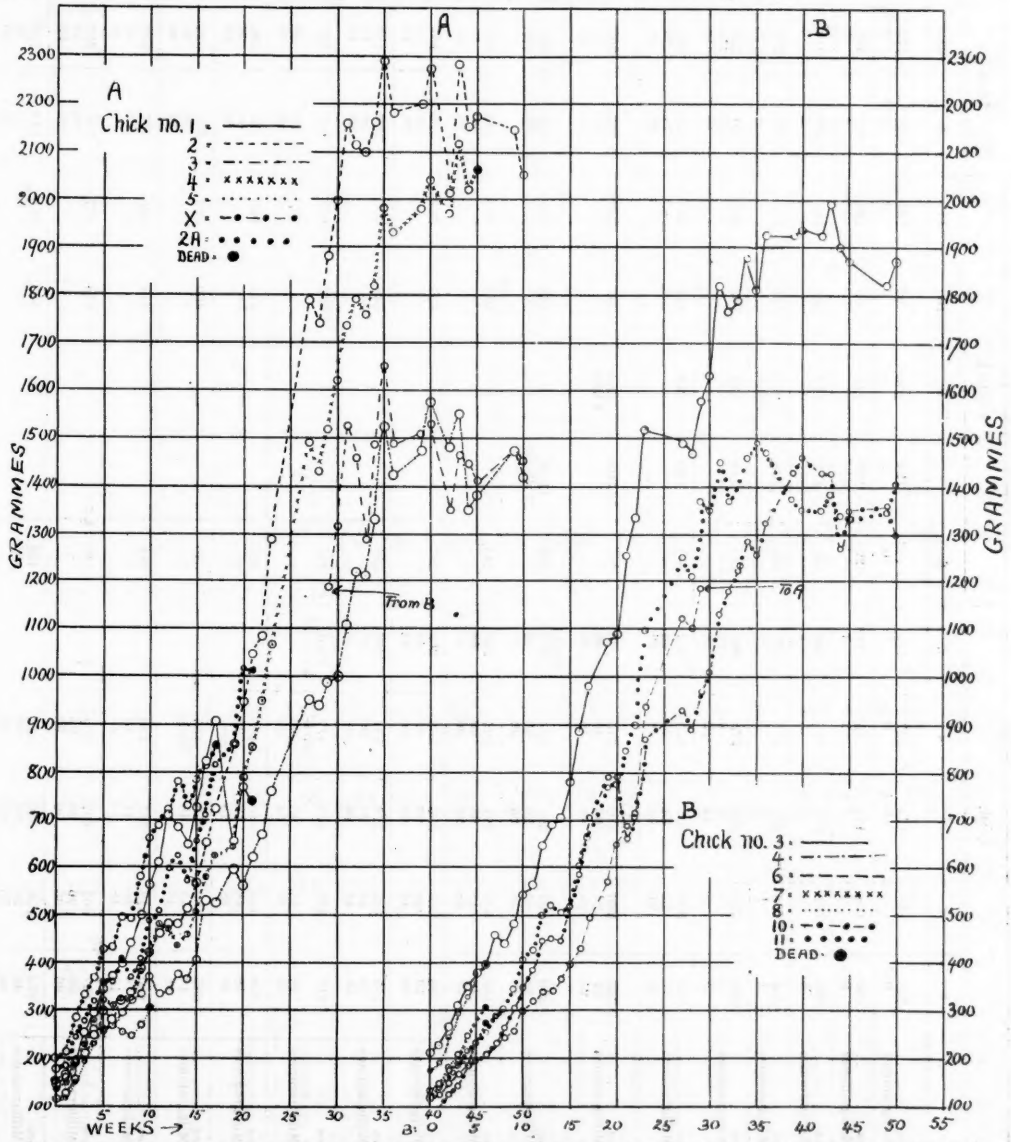
CHICKS from eggs of known lead content were fed on continually increasing amounts of colloidal lead orthophosphate. The lead content of eggs from the fowls receiving lead, and of controls, was determined, also any effect of the lead diet on the fertility of the eggs was noted. Results from these experiments are presented here.

In a previous paper(1) eggs from a fowl at Baulkham Hills were examined over a considerable period in regard to the lead content. It was shown

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney, and with the aid of the Cancer Research and Treatment Fund.

that this amount averaged 0·035 milligramme per yolk and white. Eggs from this fowl were incubated and the chicks therefrom formed the starting point for the experiments reported here.

The chicks were confined in wire cages for the first six weeks, lead as lead acetate in water being given after the third week, starting at 0·1 milligramme of lead and increasing each week. Each chick was given a different amount of lead in aqueous solution per mouth. After four weeks it was considered that the lead acetate was too toxic, so, rather than reduce the amount of lead, a change was made to the colloidal lead orthophosphate, which was being used by the Cancer Treatment Committee and which previous experiments (*loco citato*) had shown to be less toxic than the acetate. The doses of lead as the colloidal orthophosphate were increased rapidly without markedly toxic symptoms developing. The chicks were weighed before each dose and the growths plotted as shown in Graph I, which indicates that the lead had little or no adverse effects on normal growth.



Graph showing growth curves for fowls given in Table I. The graph is divided into two parts, A for fowls receiving lead, and B for control fowls.

After three months the chicks were transferred to experimental pens provided by the Department of Veterinary Science, where they lived under normal yard conditions, the controls being kept in a separate pen to those receiving the lead orthophosphate.

The weights and dosages are given in detail in Table I and summarized in Table II. It will be seen

TABLE I.

Lead given as an Aqueous Solution of Lead Acetate for Three Weeks; from then onward Colloidal Lead Orthophosphate was used.

Date.	Chicks All Six Weeks Old.											Chicks Eight Weeks Old.			
	Lead.						Control.					Lead.		Control.	
	Chick No.	1	2	3	4	5	6	7	8	10	11	X	2A	3	4
11.11.29	Weight in Grammes Dose of Lead in Milligrammes	145 0·1	148 0·2	114 0·4	137 0·8	114 1·6	118 0	121 0	130 0	92 0	120 0	200 0·5	179 0·5	211 —	173 —
18.11.29	Weight in Grammes Dose of Lead in Milligrammes	160 0·2	158 0·4	127 0·8	150 1·6	118 3	122 —	138 —	145 —	95 —	130 —	212 1	181 1	226 —	181 —
25.11.29	Weight in Grammes Dose of Lead in Milligrammes	199 0·2	192 0·4	157 0·8	192 1·6	150 3	147 —	172 —	177 —	122 —	161 —	285 2	244 2	265 —	244 —
2.12.29	Weight in Grammes Dose of Lead in Milligrammes	252 1	208 1	204 2	223 4	193 6	168 —	198 —	206 [Sick]	141 —	182 —	327 2	277 4	308 —	288 —
9.12.29 13.12.29	Weight in Grammes Dose of Lead in Milligrammes	293 3 6	248 3 6	262 6 12	278 6 12	232 12 24	185 —	248 —	182 —	182 —	215 —	367 12 12	320 12 18	350 —	327 —
16.12.29 20.12.29	Weight in Grammes Dose of Lead in Milligrammes	345 12 24	296 12 24	307 24 48	320 24 48	263 48 96	192 —	290 —	230 —	190 —	290 —	426 24 48	357 24 48	375 —	374 —
23.12.29 27.12.29	Weight in Grammes Dose of Lead in Milligrammes	367 24 24	280 24 24	[Sick]	310 48 48	270 96 96	207 —	303 —	280 Killed	207 —	258 —	432 48 48	360 48 48	394 —	395 —
30.12.29 3.1.30	Weight in Grammes Dose of Lead in Milligrammes	399 48 48	329 48 48	295 96 96	324 96 96	[Sick]	233 —	Died, 29.12.29	— —	235 —	288 —	495 96	407 200	460 —	Died, 29.12.29
6.1.30 10.1.30	Weight in Grammes Dose of Lead in Milligrammes	440 48 48	336 48 48	321 96 96	325 96 96	248 48 48	243 —			265 —	300 —	493 96 96	367 48 48	440 —	
13.1.30 17.1.30	Weight in Grammes Dose of Lead in Milligrammes	500 48 48	385 48 48	339 96 96	382 96 96	268 48 48	257 —			304 —	347 —	580 96 96	407 48 48	483 —	
20.1.30 24.1.30	Weight in Grammes Dose of Lead in Milligrammes	565 48 48	423 48 48	372 96 96	421 96 96	303 48 48	299 —			348 —	405 —	660 96 96	489 48 48	538 —	
27.1.30 31.1.30	Weight in Grammes Dose of Lead in Milligrammes	610 48 33	460 48 33	333 48 33	477 [Sick] 96 66	Died, 25.1.30	317 —			382 —	424 —	687 96 66	507 48 33	566 —	
3.2.30 7.2.30	Weight in Grammes Dose of Lead in Milligrammes	708 48 48	483 48 48	340 48 48	468 96 —		337 —			445 —	500 —	720 96 96	595 96 96	645 —	
10.2.30 14.2.30	Weight in Grammes Dose of Lead in Milligrammes	685 48 48	483 48 48	375 48 —	435 [Sick]		340 —			450 —	520 —	785 96 96	625 96 96	690 —	
17.2.30 21.2.30	Weight in Grammes Dose of Lead in Milligrammes	650 48 48	512 48 48	367 48 48	460 48 48		365 —			448 —	505 —	727 96 96	572 96 96	705 —	
24.2.30 28.2.30	Weight in Grammes Dose of Lead in Milligrammes	727 96 96	565 96 96	407 96 96	505 96 96		390			518	517	785 96 96	655 96 96	778 —	
3.3.30 7.3.30	Weight in Grammes Dose of Lead in Milligrammes	823 96 96	650 96 96	530 96 96	577 96 96		428 —			585 —	600 —	818 96 96	710 96 96	887 —	
10.3.30 14.3.30	Weight in Grammes Dose of Lead in Milligrammes	905 96 96	720 96 96	525 48 —	623 96 96		498 —			710 —	668 —	855 96 96	815 96 96	978 —	
24.3.30	Weight in Grammes Dose of Lead in Milligrammes	655 96	857 96	597 96	642 96		570 —			790 —	770 —	Died, 19.3.30	860 96	1,070 —	

24.3.30	Weight in Grammes	655	857	597	642		515			—	—		19.3.30	—	
	Dose of Lead in Milligrammes	96	96	96	96		—						96		

TABLE I (*Continued*).

31.3.30	Weight in Grammes	766	946	562	788		649			789	779		1,014	1,087	
	Dose of Lead in Milligrammes	96	96	96	96		—			—	—		96	—	
7.4.30	Weight in Grammes	740	1,042	620	851		688			660	848		1,011	1,256	
	Dose of Lead in Milligrammes	[Sick]	148	96	96		—			—	—		96	—	
14.4.30	Weight in Grammes	Died,	1,080	670	950		715			709	900		Killed,	1,333	
18.4.30	Dose of Lead in Milligrammes	2.4.30	96	46	96		—			—	—		10.4.30	—	
			52	96	96										
21.4.30	Weight in Grammes		1,288	760	1,163		938			870	1,094			1,517	
25.4.30	Dose of Lead in Milligrammes		96	96	96		—			—	—			—	
			48	96	148										
19.5.30	Weight in Grammes		1,785	950	1,490		1,120			930	1,250			1,490	
	Dose of Lead in Milligrammes		96	96	96		—			—	—			—	
			48	96	96										
26.5.30	Weight in Grammes		1,740	940	1,430		1,100			890	1,210			1,470	
30.5.30	Dose of Lead in Milligrammes		96	96	96		—			—	—			—	
			96	96	96										
2.6.30	Weight in Grammes		1,878	988	1,518		1,118			970	1,368			1,580	
6.6.30	Dose of Lead in Milligrammes		96	144	96		—			—	—			—	
			96	96	96										
9.6.30	Weight in Grammes		1,998	1,000	1,620		1,315			1,012	1,350			1,630	
13.6.30	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
			96	96	96										
16.6.30	Weight in Grammes		2,155	1,105	1,735		1,525			1,130	1,450			1,820	
20.6.30	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
			96	96	96										
23.6.30	Weight in Grammes		2,112	1,217	1,790		1,460			1,180	1,370			1,768	
27.6.30	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
			96	—	96		96								
30.6.30	Weight in Grammes		2,100	1,213	1,759		1,290			1,220	1,235			1,787	
4.7.30	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
			96	96	96		—								
7.7.30	Weight in Grammes		2,160	1,330	1,822		1,487			1,284	1,465			1,879	
11.7.30	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
			96	96	96		96								
14.7.30	Weight in Grammes		2,285	1,524	1,980		1,655			1,254	1,490			1,810	
	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
21.7.30	Weight in Grammes		2,176	1,424	1,930		1,478			1,327	1,470			1,925	
	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
11.8.30	Weight in Grammes		2,220	1,472	1,980		1,507			1,430	1,375			1,925	
	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
18.8.30	Weight in Grammes		2,272	1,575	2,040		1,530			1,462	1,353			1,935	
22.8.30	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
			44	44	44		44								
1.9.30	Weight in Grammes		2,010	1,480	1,970		1,350			1,430	1,350			1,924	
5.9.30	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
			44	44	44		44								
8.9.30	Weight in Grammes		2,280	1,550	2,115		1,465			1,433	1,385			1,990	
12.9.30	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
			96	96	96		96								
15.9.30	Weight in Grammes		2,150	1,350	2,020		1,450			1,273	1,339			1,900	
19.9.30	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
			48	96	96		—								
22.9.30	Weight in Grammes		2,170	1,380	2,060		1,410			1,350	1,330			1,870	
	Dose of Lead in Milligrammes		96	96	96		96			—	—			—	
20.10.30	Weight in Grammes		2,145	1,473	Killed,		1,470			1,360	1,345			1,820	
24.10.30	Dose of Lead in Milligrammes		96	96	24.9.30		96			—	—			—	
			96	96			96								
27.10.30	Weight in Grammes		2,050	1,418			1,451			1,300	1,404			1,870	
31.10.30	Dose of Lead in Milligrammes		96	96			96			—	—			—	
			96	96			96								

TABLE II.

Lead given as an Aqueous Solution of Lead Acetate for Three Weeks; from then onward Colloidal Lead Orthophosphate was used.

	Six Weeks Old.										Eight Weeks Old.			
	Lead.						Control.				Lead.		Control.	
Chick No.	1	2	3	4	5	6	7	8	10	11	X	2A	3	4
Age (weeks)	27⅛	56⅛	56⅛	55⅛	10⅛	56⅛	12⅛	12	56⅛	56⅛	26⅛	29⅛	56⅛	12⅛
Weight (grammes) ..	740	2,050	1,418	2,070	303	1,451	303	280	1,300	1,404	855	1,011	1,870	395
Total dose of lead in milligrammes ..	1,615	4,927	5,381	5,202	865·6	1,144	0	0	0	0	1,157	1,350	0	0
Period of dose (weeks)	21	50⅛	50⅛	49⅛	4⅛	21	—	—	—	—	18⅜	21⅜	—	—
Average weekly dose (milligrammes) ..	77	97·25	106	105	18·9	54·5	—	—	—	—	63·2	63·0	—	—
Whether still living ..	No	No	Yes	No	No	Yes	No	No	Yes	Yes	No	No	Yes	No
Cause of death ..	Cold	Cold	—	Killed for analysis	Rats	—	Cold	Rats	—	—	Rats	Rats	—	Rats

that a dose of 96 milligrammes of lead is about the maximum that can be given at one time without causing a drop in weight. Where a large falling off in weight was noticed, the dose of lead was reduced until the weight was regained. This explains the variations in the doses given from week to week.

Lead Output.

The chicks were being given lead in quite large doses. What was the fate of this lead? How much was retained by the fowl? An attempt to answer this question was made soon after the feeding was commenced. Two chicks (numbers 3 and 8), one receiving lead, the other a control, were placed in separate glass floored cages and the fæces collected daily for one week, the lead being estimated in the day's droppings. The results are shown in Table III.

The droppings from the control chick were firm, round and dark in colour, while those from the chick receiving lead were at no time firm, were without form, and white in colour.

It is apparent that, although quite a large amount of lead is rejected by the fowl, there is a "carry over" from the last dose of approximately 30% in the experiment cited.

Analysis of Fowls.

In Table I it will be noted that many of the fowls died, some from disease, others being killed by rats. In each case the remainder of the dead fowl was dissected and the lead estimated. What was left after the dissection was passed through a mincing machine and was also examined for lead. This material is placed in the column headed "Remainder" of Table IV, wherein appears the analysis of seven fowls, two being controls receiving no added lead, the other five having received lead as shown in Table I, the total dosage only being given in Table IV.

This table is particularly interesting and, while a detailed discussion is unnecessary, the following points commend themselves.

The intestine, crop and gizzard need not be considered, as one would naturally expect to find lead in these parts, and, furthermore, the bulk of the lead is still outside the body, not having been absorbed. In the normal chick we find in both instances that the brain contains most lead, then liver, bone and feathers. After the addition of lead we find that bone and feathers absorb lead far more rapidly than the liver and brain. When the ovaries appear, during laying, the amount of lead

TABLE III.

Output of Lead by Fowl after Oral Dosage of Colloidal Lead Orthophosphate.

Chick No.	Time Sample taken after Injection.	Dry Weight of Fæces, Grammes.	Milligrammes of Lead in	
			Fæces.	100 Grammes of Dry Fæces.
8 (control)	—	100	0·625	0·625
3	2 days after 48 milligrammes dose	25·0	1·33	5·32
Given 96 milligrammes lead ..	20 minutes	6·00	1·33	22·20
	24 hours (1 day)	10·00	31·60	316·0
	48 hours (2 days)	12·0	22·5	187·50
	72 hours (3 days)	9·3	7·43	79·50
	96 hours (4 days)	14·9	1·57	10·50
	120 hours (5 days)	12·8	1·05	8·20
	144 hours (6 days)	17·10	1·30	7·60
	168 hours (7 days)	8·5	0·75	8·82

Because of the fluidity of the fæces, the glass floors had to be rinsed with distilled water. This precluded a determination of the wet weight of the fæces.

TABLE IV.

Distribution of Lead in Fowls.

			Intestine.	Crop.	Gizzard (with Contents).	Liver.	Bone.	Feathers.	Muscle.	Lungs.	Brain.	Ovaries.	Blood.	Kidney.	Heart.	Remainder.
Chick No. ..	[Control] 8	Weight in Grammes of Part — Wet ..	15·50	23·50	32·50	8·50	29·00	12·00	50·30	8·57	2·50		10·25		3·75	85·00
Age in Weeks ..	12	Weight in Grammes of Part — Dry ..	4·50	13·50	12·00	2·50	10·00	6·50	12·70	0·86	0·50		2·33		1·21	29·00
Weight in Grammes	280	Water %	71·00	42·50	63·0	70·50	65·50	45·7	74·90	90·00	80·00		77·50		67·60	65·80
Days since Last Dose	—	Milligrammes of Lead in — Part ..	0·031	0·06	0·028	0·037	0·092	0·046	0·08	0·006	0·014		0·005		0·001	1·34
		Milligrammes of Lead in — 100 Grammes Wet ..	0·200	0·255	0·086	0·435	0·317	0·384	0·159	0·070	0·560		0·048		0·027	1·58
Total Lead given (in Milligrammes)	—	Milligrammes of Lead in — 100 Grammes Dry ..	0·690	0·445	0·234	1·480	0·920	0·710	0·630	0·698	2·80		0·214		0·088	4·62
Chick No. ..	[Control] 7	Weight in Grammes of Part — Wet ..	37·00	22·50	23·50	11·50	10·50	45·70	23·00	9·45	1·95		17·52	1·72	4·23	94·50
Age in Weeks ..	12⁴/₇	Weight in Grammes of Part — Dry ..	5·00	6·00	12·00	2·50	4·00	11·40	6·00	0·88	0·24		3·57	0·52	1·05	31·62
Weight in Grammes	303	Water %	86·40	73·50	49·0	78·30	61·90	75·00	74·00	90·80	87·60		79·50	69·80	75·10	66·50
Days since Last Dose	—	Milligrammes of Lead in — Part ..	0·121	0·034	0·043	0·031	0·113	0·09	0·046	0·007	0·007		0·001	Less than 0·001	Less than 0·001	1·02
		Milligrammes of Lead in — 100 Grammes Wet ..	0·327	0·151	0·183	0·270	1·08	0·197	0·200	0·074	0·359		0·006	Less than 0·058	Less than 0·024	1·08
Total Lead given (in Milligrammes)	—	Milligrammes of Lead in — 100 Grammes Dry ..	2·420	0·567	0·360	1·240	2·82	0·790	0·766	0·795	2·92		0·028	Less than 0·190	Less than 0·095	3·22
Chick No. ..	5	Weight in Grammes of Part — Wet ..	18·62	16·12	50·15	6·60	11·56	50·00	16·88	11·40			8·77			113·50
Age in Weeks ..	10⁶/₇	Weight in Grammes of Part — Dry ..	4·79	4·02	22·05	1·41	2·84	20·00	3·41	1·00			1·80			43·00
Weight in Grammes	303	Water %	74·0	75·00	56·00	78·60	75·50	60·00	79·60	91·20			79·50			62·10
Days since Last Dose	5	Milligrammes of Lead in — Part ..	7·00	0·10	1·17	0·10	1·60	1·28	0·049	0·012			0·015			10·00
		Milligrammes of Lead in — 100 Grammes Wet ..	37·6	0·620	2·33	1·51	13·85	2·56	0·291	0·105			0·171			8·80
Total Lead given (in Milligrammes)	865·6	Milligrammes of Lead in — 100 Grammes Dry ..	146·00	2·480	5·30	7·08	56·40	6·40	1·44	1·20			0·835			23·30
Chick No. ..	X	Weight in Grammes of Part — Wet ..	57·50	31·00	124·30	16·46	13·55	2·75	17·91	18·54	2·30			2·42		568·5
Age in Weeks ..	26⁵/₇	Weight in Grammes of Part — Dry ..	11·50	9·00	29·30	2·96	3·55	0·60	1·91	2·44	0·36			0·94		220·0
Weight in Grammes	855	Water %	80·00	70·80	76·50	82·00	73·80	78·15	89·10	86·60	84·30			61·20		61·40
Days since Last Dose	9	Milligrammes of Lead in — Part ..	5·33	8·05	4·72	0·20	1·43	0·14	0·05	0·063	0·02			0·005		65·0
		Milligrammes of Lead in — 100 Grammes Wet ..	9·26	26·00	3·80	1·22	10·60	5·10	0·28	0·339	0·870			0·206		11·41
Total Lead given (in Milligrammes)	1,157	Milligrammes of Lead in — 100 Grammes Dry ..	46·40	89·50	16·10	6·75	40·25	23·40	2·62	2·580	5·550			0·532		29·60
Chick No. ..	2A	Weight in Grammes of Part — Wet ..	57·91		91·00	39·96	11·20	9·45	50·30	17·00		1·20		2·08	7·00	514
Age in Weeks ..	29⁸/₇	Weight in Grammes of Part — Dry ..	10·61	Neck, Leg, Wing, and Crop Eaten by Rats	45·50	9·00	3·20	2·35	11·84	1·20		0·20		0·85	2·30	200
Weight in Grammes	1,011	Water %	81·70		50·00	77·50	71·40	75·0	76·50	93·00		83·50		59·10	67·20	61·30
Days since Last Dose	3	Milligrammes of Lead in — Part ..	2·33		3·19	0·57	5·00	0·318	0·116	0·070		0·152		0·012	0·054	69·70
		Milligrammes of Lead in — 100 Grammes Wet ..	4·03		3·51	1·43	44·60	3·37	0·231	0·412		12·70		0·577	0·771	13·60
Total Lead given (in Milligrammes)	1,350	Milligrammes of Lead in — 100 Grammes Dry ..	21·90		7·02	6·35	156·20	13·55	0·980	5·84		76·00		1·41	2·35	34·85

TABLE IV (*Continued*).

				Intestine.	Crop.	Gizzard (with Contents).	Liver.	Bone.	Feathers.	Muscle.	Lungs.	Brain.	Ovaries.	Blood.	Kidney.	Heart.	Remainder.
Chick No... ..	1	Weight in Grammes of Part	Wet	45·00	16·00	35·00	20·00	14·00	3·00	13·00	15·00	2·00	0·90	12·00	2·03	4·00	391·00
Age in Weeks ..	27½		Dry	6·00	5·30	11·70	5·50	5·00	1·60	3·40	2·10	0·40	0·10	2·20	0·74	0·50	143·3
Weight in Grammes	740	Water %		86·50	67·00	66·50	72·50	64·30	46·80	74·00	86·00	80·00	89·00	81·60	63·50	87·50	63·2
Days since Last Dose	12	Milligrammes of Lead in	Part 100 Grammes Wet	0·28	0·20	0·44	0·41	5·20	0·33	0·23	0·09	0·08	0·27	0·126	0·018	0·009	81·15
Total Lead given (in Milligrammes)	1,015		100 Grammes Dry ..	0·624	1·25	1·26	2·05	37·20	11·10	1·77	0·60	4·00	30·00	1·05	0·884	0·225	20·8
				4·060	3·78	3·76	7·45	104·00	20·60	6·75	4·28	20·00	270·00	5·73	2·44	1·80	57·0
Chick No... ..	2	Weight in Grammes of Part	Wet	23·63	34·52	29·23	21·28	66·79	11·35	37·50	35·70	2·54	1·99	25·42	22·91	14·15	1,790
Age in Weeks ..	62½		Dry	6·10	8·72	8·99	6·10	21·96	5·67	11·63	4·13	0·53	0·23	4·92	7·01	2·51	645
Weight in Grammes	2,100	Water %		78·10	75·00	69·00	70·50	67·00	50·00	69·00	88·45	79·00	88·50	80·60	65·50	82·00	64·0
Days since Last Dose	5	Milligrammes of Lead in	Part 100 Grammes Wet	0·16	0·292	0·20	0·35	15·00	0·28	0·029	0·24	0·102	0·42	0·274	0·025	0·01	544
Total Lead given (in Milligrammes)	5,381		100 Grammes Dry ..	0·678	0·845	0·68	0·164	22·50	2·47	0·077	0·672	4·02	21·1	1·08	0·109	0·071	30·4
				2·62	3·35	2·23	5·74	68·40	4·95	0·249	5·81	19·20	182·5	5·56	0·316	0·398	84·5
Chick No... ..	4	Weight in Grammes of Part	Wet	99·45	148·80	54·50	49·00	71·40	8·50	40·80	30·64	2·06		5·70	25·65	11·90	1,569
Age in Weeks ..	55½		Dry	18·45	74·9	11·60	13·90	34·3	2·52	10·20	3·84	0·31		1·50	5·65	3·20	629
Weight in Grammes	2,070	Water %		81·5	49·6	78·50	71·50	52·0	70·25	75·00	87·5	85·0		73·5	78·0	73·20	60·0
Days since Last Dose	1	Milligrammes of Lead in	Part 100 Grammes Wet	0·30	0·30	0·143	0·077	4·50	0·135	0·03	0·10	0·08		0·005	0·29	0·056	432
Total Lead given (in Milligrammes)	5,202		100 Grammes Dry ..	0·302	0·202	0·262	0·157	6·30	1·59	0·073	0·326	3·88		0·088	1·13	0·470	27·50
				1·63	0·400	1·23	0·555	13·10	5·35	0·294	2·60	25·80		3·34	5·15	1·75	68·50

absorbed by them is quite large and is of the same order as the bone.

It is rather striking that the content of lead in the muscle varies but slightly, almost remaining unchanged, even after prolonged dosage with lead. The blood, on the other hand, does show an increase in lead content.

Chick number 2 shows that the lead is being retained in the body, for, with an interval of five weeks after the last dose of lead, we find comparatively small amounts in the crop, intestine and gizzard, as we might expect. The lead content of the bone, ovaries and brain are still high, although that of the feathers is very low. It is interesting to observe that the lead content of the heart and muscle is approximately the same, and that for the muscle it is lower than that for the normal chick.

Table V shows the actual amount of lead found in the fowls, both for controls and those dosed with lead, and shows clearly that lead is absorbed after long treatment. The high concentration of lead in the ovaries leads one to expect an absorption of lead by the egg. Thus we might expect to find an increase in the lead content of those eggs from the hens being dosed with lead. A definite increase is found in the eggs, as is shown in Table VI, but it does not seem possible to increase the amount beyond a limit which appears to be fixed by the normal lead content of the egg, and as has been shown in an earlier paper may vary between wide limits.

The normal lead content of the eggs from which these fowls were hatched was found to be about 0·035 milligramme. The lead content of eggs from chicks hatched from these eggs is not at any time largely in excess of this figure. The lead content of eggs from the chicks dosed with lead may rise to five times this amount.

The fowls receiving lead laid just as well as those without lead. In the beginning those receiving the lead began laying before those without lead and laid more eggs than those without lead. Since it was found that the lead content of the eggs could be raised by prolonged feeding of the fowls with lead added to their food, it remained to be seen whether the fertility of the eggs was affected in any way.

The whites of the eggs from the leaded fowls were generally much more fluid than those from the controls. A determination of the pH (hydrogen ion) concentration, using a glass electrode, showed no difference between the two.

TABLE V.

Total Lead in Normal and Leaded Fowls.

Chick No.	Age in Weeks.	Weight, Grammes.	Total Dosage.	Lead in Milligrammes.		Percentage of Lead Retained by Fowl.
				Found per Chick.	Found per 100 Grammes Wet Material.	
8 (control)	12	280	—	1·75	0·625	—
7 (control)	12⁶/₇	303	—	1·51	0·500	—
5	10⁶/₇	303	865·5	21·3	7·02	2·46
X	26⁶/₇	855	1,157	85·04	9·95	7·37
2A (part of chick eaten by rats)	29⁵/₇	1,011	1,350	81·51	8·05	6·05
1	27⁴/₇	740	1,615	88·83	12·0	5·50
2	62⁴/₇	2,100	5,381	561·98	26·8	10·45

Table V shows the actual amount of lead found in the fowls, both for controls and those dosed with lead, and shows clearly that lead is absorbed after long treatment.

TABLE VI.

Eggs of Fowls Hatched from Eggs from Baulkham Hills.
The Original Eggs had been found to have an Average Lead Content of 0·035 Milligramme of Lead per Egg [1, loco citato].

Egg Laid on	Weight of Egg (Grammes).	Wet Weight in Grammes of			Milligrammes of Lead found in			Milligrammes of Lead per 100 Grammes of Wet.		
		Shell.	Yolk.	White.	Shell.	Yolk.	White.	Shell.	Yolk.	White.
4.6.30	47·0085	5·6047	14·2542	27·1496	0·014	0·037	0·019	0·250	0·260	0·070
11.6.30	43·5440	5·3172	14·1692	24·0576	0·007	0·025	0·034	0·130	0·175	0·141
16.6.30	48·2070	6·3408	15·7182	26·1480	0·016	0·021	0·006	0·250	0·130	0·023
18.6.30	46·6622	5·8044	14·8752	25·9826	0·014	0·016	0·019	0·241	0·108	0·073
19.6.30	51·2529	7·2048	15·5826	28·4655	0·013	0·024	0·019	0·180	0·154	0·067
24.6.30	45·8312	6·2544	17·9175	21·6593	0·009	0·010	0·005	0·144	0·056	0·023
26.6.30	49·2948	6·5126	19·3244	23·4578	0·010	0·017	0·017	0·154	0·088	0·073
27.6.30	44·6826	6·3756	17·2570	21·0500	0·014	0·028	0·014	0·220	0·162	0·066
1.7.30	44·7543	7·2540	18·3078	19·1925	0·010	0·013	0·010	0·138	0·071	0·052
2.7.30	48·0987	6·8055	18·8176	22·4756	0·006	0·020	0·007	0·088	0·106	0·031
14.8.30	45·4876	6·5574	14·4426	24·4856	0·015	0·010	0·007	0·229	0·069	0·028
18.8.30	50·5009	6·7740	16·5975	27·1294	0·009	0·034	0·026	0·130	0·206	0·096
Eggs of Fowls given Lead Orthophosphate.										
17.6.30	48·3666	6·3045	15·5076	26·5545	0·029	0·045	0·026	0·460	0·290	0·098
18.6.30	50·4641	6·7012	16·1542	27·6087	0·017	0·017	0·043	0·25	0·105	0·155
19.6.30	44·3659	6·2078	13·0035	25·1546	0·025	0·021	0·044	0·405	0·161	0·174
24.6.30	49·9137	6·8546	17·2570	25·8021	0·023	0·067	0·031	0·336	0·389	0·120
1.7.30	55·7012	7·2004	17·8576	30·6432	0·040	0·029	0·029	0·555	0·163	0·095
2.7.30	51·2643	7·5577	17·0952	26·6114	0·020	0·043	0·043	0·266	0·252	0·162
3.7.30	52·4619	7·1564	18·0543	27·2512	0·020	0·041	0·034	0·280	0·226	0·124
6.7.30	51·0106	6·1048	18·8047	26·1011	0·043	0·043	0·050	0·705	0·228	0·192
9.7.30	46·8198	5·9542	17·7100	23·1556	0·016	0·057	0·058	0·269	0·322	0·251
10.7.30	43·0821	5·8172	13·6544	23·6105	0·008	0·029	0·029	0·138	0·212	0·123
18.8.30	47·1565	6·7520	15·3501	25·0544	0·024	0·027	0·026	0·356	0·176	0·104
20.8.30	56·7359	7·3561	18·3752	31·0046	0·044	0·046	0·035	0·598	0·250	0·113

Summary.

Eggs from	Average Lead Content in Milligrammes of			Total Lead per Egg.
	Shell.	Yolk.	White.	
Control Hens	0·011	0·021	0·015	0·047
Leaded Hens	0·026	0·039	0·035	0·090

When the eggs were incubated, interesting differences were apparent, as will be seen in Table VII. Generally equal numbers of both series of eggs were incubated together, so as to eliminate any differences due to incubation conditions. The difference between the control eggs and the leaded eggs may be due to the following: (i) The leaded hens have become sterile, (ii) the leaded rooster is sterile, (iii) pathological conditions of the testes and ovaries of the birds were present.

To try to clear this point the roosters were changed over, the leaded rooster now being with the control hens, while the control rooster is with the leaded hens. The hens were left without the rooster until all eggs laid were sterile; then the exchange was made. After allowing one month to elapse since making the exchange, the eggs were incubated. The results appear in Table VIII and are summarized in Table IX.

It is not possible to draw any conclusions from these results for the reason that the percentage of hatched chicks from the control (normal) fowls is so low and also for the reason that the number of eggs from the hens after the exchange of roosters as given in Table VIII is relatively small. Work is still being continued on this part of the investiga-

TABLE VII.

Eggs from the Control and Leaded Hens Incubated under Usual Conditions of Temperature and Humidity in a "Buffalo" Incubator for Twenty-one Days.

Date.	Eggs.	Number Incubated.	Results.		
			Sterile.	Dead Chick.	Chick Alive.
25.7.30 to 23.10.30	Control	68	6	26	36
25.7.30 to 20.10.30	Leads	64	42	14	8

TABLE VIII.

Roosters Exchanged. Eggs Incubated under the Same Conditions as in Table VII.

Date.	Eggs.	Number Incubated.	Results.		
			Sterile.	Dead Chick.	Chick Alive.
1.12.30 to 15.12.30	Leaded rooster and control hens	33	6	0	27
1.12.30 to 15.12.30	Control rooster and leaded hens	45	12	18	15

TABLE IX.

Type of Bird.	Percentage of Eggs.		
	Sterile.	Dead.	Alive.
Leaded rooster and leaded hens { Table VII }	65·6	21·85	12·50
Control rooster and hens .. { Table VII }	8·82	38·20	53·00
Leaded rooster and control hens { Table VIII }	18·20	—	81·80
Control rooster and leaded hens { Table VIII }	26·70	40·00	33·3

tion, when it is felt that larger numbers of eggs will yield results of some value.

With regard to the low percentage of hatched chicks from the control eggs, the State Department of Agriculture gives figures for the period of the year when these eggs were incubated of 60% to 70%. Thus the figure of 53% hatched chicks is not of much value for comparison, as it is well below normal and gives a false value to the differences shown on the transfer of the roosters.

If we consider the total percentage of dead and alive chicks in all the eggs we have figures expressing results which are more nearly those we might expect. Thus we would find that the eggs from the leaded fowls have the lowest value, the normal eggs highest, while transfer of the roosters produced a nearly even state of affairs.

However, as stated above, these figures do not allow of any conclusions being drawn that might not be refuted when larger numbers of eggs and hens are considered.

Summary.

The results given in this paper show that:

1. By feeding colloidal lead orthophosphate to fowls the lead content of the fowls is raised.

2. The feeding of the lead orthophosphate does not appear to affect the growth of the fowls when kept below the amount causing toxic symptoms to appear.

3. The lead content of the eggs from fowls receiving lead is raised, but the laying capacity of the hens is not affected.

4. The eggs from the leaded fowls show a greater percentage sterility than those from the normal fowls.

Acknowledgement.

I have to thank Professor Stewart, of the Veterinary Science School for making available the experimental houses in which the fowls have been kept during these experiments. Also I wish to thank Mr. Carne, B.V.Sc., Pathologist of the Veterinary Science School, for his many helpful suggestions in the care of the fowls and for his trouble in performing *post mortem* examinations when necessary.

Reference.

(1) W. B. S. Bishop: "The Occurrence of Lead in the Egg of the Domestic Hen," Part II, THE MEDICAL JOURNAL OF AUSTRALIA, January 26, 1929; Part III, THE MEDICAL JOURNAL OF AUSTRALIA, June 15, 1929.

Reports of Cases.

TRAUMATIC RETROPERITONEAL HÆMORRHAGE.

By M. P. SUSMAN, M.B., Ch.M. (Sydney), F.R.C.S. (England),
Sydney.

A YOUTH, aged nineteen years, was thrown from the seat of his bicycle against the handle bar and he was admitted to a suburban hospital complaining of abdominal pain. He stated that he was discharged after ten days and that he still had abdominal pain so that he had to walk "doubled up." Three days later, that is, thirteen days after the accident, he had a shivering attack followed by vomiting; he had repeated bouts of shivering and vomiting during the next two days. When I saw him on the sixteenth day after the accident, he was gravely ill; his temperature was 40° C. (104° F.), his pulse rate 140 and his respiration rate 36 per minute; there was moderate uniform distension of the abdomen with but slight tenderness and rigidity; upon rectal examination he complained of tenderness anteriorly.

I opened the abdomen through a right paramedian incision; no intraperitoneal mischief was found, but there was a soft swelling in front of the vertebral column behind the peritoneum; this proved to be a collection of stinking, decomposing blood, and I drained it. The patient died a few hours later.

Post Mortem Findings.

The presence of a suppurating retroperitoneal hæmatoma was confirmed. The source of the bleeding was not found, nor was the source of infection, all the viscera and their blood supply being intact.

Comment.

Traumatic retroperitoneal hæmorrhage without gross damage to one of the retroperitoneal organs or one of the larger blood vessels is rare. Conservative treatment is usually warranted, but observation for complications must be part of this treatment. Certainly one should not dismiss a patient who has had an abdominal injury while he is still complaining of abdominal pain.

MEDIASTINAL EMPHYSEMA.

By A. M. WATKINS, M.B., B.S.,
Roseville, New South Wales.

A MAN, aged sixty-five years, consulted me for the reason that he had retired from farming and that he wished me to attend him and to advise him as to his future length of time upon the earth. He was a dairy farmer with a long grey beard and moustache, with a well preserved head of hair and a clear, decided pink colour on both cheeks and lips. If his beard and moustache had not been present, one would have made a mistake about his age.

His pulse rate was 90 per minute, no dyspnœa was present, the apex beat was in the sixth interspace just outside the nipple line, and was visible. A definite mitral regurgitant murmur and an aortic stenotic murmur were present, but over the whole precordium crepitant râles occurring during both systole and diastole of the heart were present, resembling for all the world the rattle of dried peas on taut canvas.

I diagnosed mediastinal emphysema, dismissing all thoughts of a pericardial involvement.

It is just twenty years ago, when I was clinical clerk to Sir Henry Maudsley in the Melbourne Hospital, that I was the fortunate hearer of a similar condition; although I have been a busy practitioner, I have never been granted an opportunity of hearing what I had listened to when just a boy and at that time was never diagnosed.

This man would come with me and would be quite willing to demonstrate his lesion. I could not possibly let the occasion slip by unnoticed of recording the phenomenon.

Reviews.

OBSTETRICS.

"THE THEORY OF OBSTETRICS," by M. C. De Garis, is divided into three parts.[1] Part I deals with "A New Theory of Obstetrics" and some of the subjects discussed are: healthy motherhood; cause of pain in labour; clinical studies in obstetrics; symptomatology of labour; pain and other reflexes in labour; viscero-motor reflexes in labour; uterine function in everyday labour, in persistent occipito-posterior delivery, in painless labours; uterine inertia; puerperal sepsis; diet and hidden dental sepsis and child bearing. Part II is a critical analysis of one hundred consecutive labours in her own practice. Part III deals with the application of the theory to practice.

Several of the chapters were written for special occasions and had been published in various journals, and so there is a considerable amount of repetition of various arguments. The motive of this may be, as one orator maintains, that more people grasp the idea of a fact each time it is repeated.

Dr. De Garis thinks that the reason why the maternal and infantile death rates are not falling is that there is an incomplete survey of the question at issue, and she considers a distinction should be drawn between the clinical and preventive aspects of the problem. She strongly rebuts the charge that the general practitioner is to blame. Normal labour is defined as "a labour in which the uterine contractions act thoroughly efficiently, leading in a short time to the spontaneous delivery of a healthy baby, and causing but little or no distress or suffering to the mother." Pain in every other function but labour is accepted as due to some fault in the process. Normal labour, therefore, should be painless. She considers the uterine action is the most important factor in abnormal labour in Australia, pelvic contraction being rare in her experience, and a research into the cause of painful uterine contractions during labour would, she thinks, solve many present day obstetric problems. This research should also include an inquiry into the problems affecting maternal health, for figures published show a correlation between maternal health, uterine efficiency and infantile health. The problems to be studied should be the effects of faulty diets on child bearing, the effects of septic foci, the prevalence of renal and dental disease in mothers and their danger to infants, in short, a survey of the whole maternal life history in its relation to child bearing. She herself lays great stress on the dangers of hidden dental disease.

We welcome this book as a useful addition to obstetric literature, because it calls attention to an aspect of obstetrics which hitherto has not received the attention which its importance demands, and a study of which should yield much valuable information. The book is a call to the general practitioner to record, analyse, and think about his cases.

GENERAL NURSING.

IT is only after perusing a text book such as "A Handbook for Nurses," by Dr. J. K. Watson that one begins to realize fully the wide knowledge demanded of the present day nurse.[2] In contrast with the medical student, who is perhaps overloaded with theory, the probationer nurse finds herself doing things first and possibly learning why afterwards. It may be that some day, with the establish-

[1] "The Theory of Obstetrics," by M. C. De Garis, M.D.; 1930. London: Baillière, Tindall and Cox. Demy 8vo., pp. 284. Price: 12s. 6d. net.

[2] "A Handbook for Nurses," by J. K. Watson, M.D.; Ninth Revised Edition; 1931. Crown 8vo., pp. 1067, with illustrations. London: Faber and Faber; Sydney: Angus and Robertson.

ment of degrees and higher degrees in nursing, the theoretical part of her training may be offered to her less haphazardly; meantime, while she has to depend on lectures of varying brilliance by teachers whose enthusiasm may be slight, good text books may help her to overcome any disadvantage due to her fortuitous choice of a training school. Dr. Watson has written a clear and comprehensive book and in this edition (the ninth) he has had the assistance of three of the leaders of the nursing profession in England. Every department of general nursing is dealt with, and the questions set by the General Nursing Council for England and Wales at the last examination are given in the appendix. The problem of condensing a text book into a hand book has been met by the use of slightly smaller print and by adopting marginal notes instead of headings. Though the effect is to make the reading appear heavy, the author's style is very simple and lucid, and the book will be found less tiring than those with the type variations, headings and subheadings so popular with some teachers.

HISTOLOGY.

LEWIS AND STÖHR'S "Histology" needs no special introduction to histologists, as it has ranked as one of the standard text books in the English language now for a number of years.[1] This new edition has been edited again by Professor Lewis's colleague, J. L. Bremer, who was also responsible for the third edition. This edition differs from the last in that less space has been given to the histology of the nervous system and to microscopical technique. It was thought desirable not to overburden an elementary text book such as this by giving a too detailed account of neuro-histology and of technical methods, as so many excellent works on these subjects are now available. The subject matter is thus restricted to what is usually undertaken in a modern course of general histology.

Professor Lewis's original plan of introducing the study of the adult structure by a description of the embryological development has been followed as closely as possible, and thus the maximum correlation of histology with embryology has been obtained. This has always been one of the most useful features of this text book.

The subject matter generally has been brought up to date, especially in the section dealing with the connective tissues and the blood, with the nervous tissue, liver and the development of the heart. Attention has also been drawn to the more important of the original contributions in the various sections.

With a few exceptions the illustrations are clear and generally help to elucidate the text. Many new illustrations are noticed, and some of the old ones have been discarded. It is particularly pleasing to notice that the original concise presentation has been preserved.

Altogether the work continues to be a creditable production, and ought to maintain its position as one of the most important text books in this subject.

RECENT WORK IN THERAPEUTICS.

"GENERAL THERAPEUTICS," by Dr. Bernard Fantus, contains the records of the therapeutic successes (and the failures) for the past year.[2] It is one of eight volumes that make up the "Practical Medicine Series," published from Chicago. The volume commences by deploring the squeezing out of therapeutics in general, and the art of prescribing in particular, from the curriculum of most modern medical schools. During the last ten years the advances in medicine have been mostly on the scientific side. It is therefore not a matter for great astonishment that the empirical and very practical side of the healing art has been more or less neglected. This anomaly will surely be readjusted, especially when those in charge of medical education realize that, as well as their function of directing, inspiring and personally conducting research, they are also obliged to turn out from their schools tolerably efficient workmen.

The author considers therapeutics under the heading of technic (that blessed word), antipathogens (whatever they are), tissue alterants, function modifiers, toxicology and physical agents. There are a few grains of wheat in the volume, but the chaff makes them very difficult to find. For the practitioner who does his fair share of reading, the book, even at the very moment of its birth, is already out of date. For those who prefer to gain their knowledge of medical progress in tablet form, the English "Medical Annual" is infinitely superior in subject matter, lucidity, intelligent commentary and general reproduction. We are unable to recommend this volume to our readers.

[1] "A Text-book of Histology Arranged upon an Embryological Basis," by J. Lewis Bremer, M.D. (Fourth Edition of "Lewis and Stöhr"); 1930. Philadelphia: P. Blakiston's Son and Company. Royal 8vo., pp. 580, with 486 illustrations, 32 of which are in colour.

[2] The Practical Medicine Series: "General Therapeutics"; Series 1930. Chicago: The Year Book Publishers. Crown 8vo., pp. 456. Price: $2.25 net.

Analytical Department.

THYREOID EXTRACT.

THE thyreoid extract produced by Glandular Preparations, Limited, Sydney, is prepared from selected thyreoid glands of Australian sheep. After removal from the carcasses the glands are conveyed to the firm's laboratories and processed the same day. They are cleaned of excess fat and connective tissue by decapsulation, are minced and desiccated by the use of acetone. Before being mixed with the excipient of the pill the desiccated mass is dried to constant weight at 37° C. and ground to a powder. This dried powder is approximately equivalent to one-fifth of the original weight of fresh thyreoid gland, so that one-fifth of a grain is the equivalent of one grain of fresh gland. This conforms to the usual practice of manufacturers of thyreoid extract. The desiccation is carried out in a cleanly manner, and the final product is uniform in appearance and free from coarse particles.

Standardization. According to a report from the Chief Veterinary Officer of the Commonwealth, Australian sheep do not suffer from goître, and the thyreoid extract from a goître-free source has much to commend it. In order that their product shall be standardized and of uniform potency, it is intended to test each batch of thyreoid extract on myxœdematous patients before releasing the batch for sale. As far as possible, the same patients will be used, and the metabolic rate will serve as a control of potency. In this way it should be possible to determine any variation in thyroxin content of the glands and adjustments will be made so that the extract as purchased will always contain the same quantity of potent thyroxin. This method of standardization is a definite advance in accuracy on that more usually employed of giving the iodine content of the gland, because the work of Harington has shown that thyroxin content and iodine content are not necessarily proportional.

In a series of patients so far tested it is found that a daily dose of one grain of thyreoid (= one-fifth of a grain *thyreoideum siccum*) raises the metabolism of one square metre of body surface four to five calories. This is in keeping with the strength of other potent brands of thyreoid extract as marketed.

The iodine content of each batch will also be estimated. So far the iodine percentage has been well over 0·3% for the desiccated gland or over 0·06% for the fresh gland. The figures ranged between 0·446% iodine and 0·434% iodine for the dried gland. Portions of the same batch have been analysed before and after processing, and it has been definitely determined that there is no loss of iodine during processing.

The product will be marketed in tablets of the usual dosages.

This attempt of an all-Australian firm to be pioneers in providing a scientifically standardized thyreoid product of known potency should commend itself to the medical practitioner.

The Medical Journal of Australia

SATURDAY, AUGUST 1, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

CONSERVATISM IN GYNÆCOLOGICAL PRACTICE.

SOME years ago a clever skit was published in one of the Australian medical students' journals on the supposed journeyings of an unfortunate sufferer from a mysterious ailment from one specialist to another. Each specialist in turn attributed the symptoms to a dysfunction of the system of which he possessed special knowledge. The patient was reminiscent of the woman in the Scriptures who "had an issue of blood twelve years, and had suffered many things of many physicians, and had spent all that she had and was nothing bettered, but rather grew worse." The fate of the patient in the story does not matter, but it served as a useful lesson to the student readers. It might be told with advantage to medical graduates. Specialism is attractive to medical students, it is the aim of many a general practitioner, and when followed in the true Hippocratic spirit, is unquestionably of most benefit to the patient. Both specialists and general practitioners, therefore, will be well advised to ask themselves occasionally whether their outlook is sufficiently broad. A recent reviewer of Morris Fishbein's "Doctors and Specialists," writing in another journal, suggested that "psychiatrists would become more like their brother practitioners if they were called upon once every two or three years to spend several weeks in the outdoor practice of an obstetric hospital," and made a somewhat similar suggestion in regard to oto-rhino-laryngologists. Such suggestions are particularly significant at the present time, when so many specialists are specialists *ab initio*, as it were, without having served an apprenticeship in general practice. Specialists who carry out most of their treatment by the knife need to be on the watch more than others. Surgical technique has become so standardized and asepsis is so easy of attainment that many procedures may be attempted by the tiro with impunity. To the skilled specialist the short cut to "cure by mutilation" is tempting, and operations may be unnecessarily extensive.

Professor W. Blair Bell has recently delivered an important address on conservative gynæcological surgery. The substance of the address has been published in *The British Medical Journal* of April 18, 1931. This paper is so pregnant with wisdom and sanity that it should be studied by all who claim a first hand surgical acquaintance with the female pelvic organs. Moreover, the principles enunciated by Professor Blair Bell, however well known they may be, may be recalled with advantage by all medical practitioners. There are three statements in the address to which we wish to call particular attention. The first is that it is curious that there should be any controversy on the question of conservatism in gynæcological surgical procedures, because the principle underlying all surgical operations is the same: "the mechanical treatment of lesions causing symptoms with the conservation of structure and function, so long as this practice does not endanger the life of the patient." The second statement is that in which he draws attention to the fact that from birth till after the menopause the genital secretions and functions play a very important part in determining the mental and physical development of the individual and in maintaining the attributes of the normal woman; and also that the ovaries are but one link in the chain of organs, such as the pituitary and thyreoid, which form the genital system. The third statement is that, as all surgeons have acquired a physiological in place of a purely anatomical outlook, so the

scientific gynæcologist sets before him the ideal of preservation of function. Conservative gynæcological surgery is thus defined by Professor Blair Bell as being concerned with the maintenance of the ovarian function and menstruation and with the fostering of conception and child-birth.

Surgeons are probably tired of being told that they are, or should be, "physicians condemned to practise surgery." Most of them realize it, if they are critical of their results. The public does not realize it; the surgeon to them is a doctor who operates, and a gynæcologist a surgeon who operates on the pelvic organs of women. The gynæcologist probably numbers among his patients more of the type of woman who "enjoys bad health" than any other specialist. Thus the temptation to discard conservatism is often great; if every possible pelvic organ is removed, the patient may then be handed over to the medical psychologist or the psychiatrist. It were better to conserve function and seek the aid of the psychiatrist in the first instance. This is often the problem of the specialist; he has intimate knowledge of the whole problem and must force himself to face it. Short reference must be made to another type of practitioner—the occasional operator (he is not to be called surgeon). This is the type of practitioner who most frequently lacks judgement; he has no intimate knowledge of the whole problem; he cannot therefore face all the facts; he is more likely to carry out unnecessary ablation and to destroy function. For both these types Professor Blair Bell has a message. In proportion as messages of this kind are received and assimilated will the outlook be made broader and the patients who "suffer many things of many physicians" be fewer.

Current Comment.

THE BLOOD PLATELETS.

THE physiological importance of blood platelets and the pathological significance of alterations in their size, numbers and appearances are not understood; their morphological characteristics are in doubt and there have been numerous theories concerning their place and mode of origin. Some investigators have expressed the opinion that they are merely precipitates from the blood plasma, others that they are fragments of leucocytes, others again that they constitute a third type of formed element of the blood. Schilling-Torgau, whose views concerning the morphology of the erythrocytes are unusual, propounded the ingenious theory that platelets are the extruded nuclei of these cells. Wright's theory that blood platelets are extruded portions of giant cells of spleen and bone marrow is the most logical and probably has the most adherents.

Though there is some difficulty in making an accurate estimation of the numbers of platelets in the blood, there is no doubt that certain diseases are often characterized by definite alterations in the numbers; it has even been suggested that disease may actually be due to such alterations.

W. Mackay has investigated the morphology and importance of blood platelets.[1] According to him the normal blood platelet is a non-nucleated body which has its origin from the megakaryocytes of the bone marrow. It has a hyaline cytoplasm containing granules; it is two to three micromillimetres in diameter and is a circular or oval disk. The nuclear appearance often observed is due to an aggregation of granules. Mackay found that the average number of platelets per cubic millimetre in health was 390,000, but he does not regard as abnormal counts of 250,000 or 450,000 per cubic millimetre. He suggests that, from the aptitude of the platelet to adhere to injured vascular epithelium and to foreign bodies in the blood and its apparent fragility outside the body, its life is probably short. It is not to be wondered at, therefore, that its numbers in the peripheral blood should vary greatly from day to day.

Generally it may be taken that the blood platelets increase in numbers during the acute phase of an infective process and decrease during convalescence. There is an increase in the numbers of platelets following hæmorrhage and a decrease associated with such disorders of the blood-forming organs as acute myeloblastic leuchæmia, chronic myeloid leuchæmia and *polycythæmia vera*. These phenomena may all be readily explained by the variations which occur in the activities of the hæmopoietic system. The condition of the platelets, both as regards quantity and morphology in relation to *purpura hæmorrhagica*, has received a good deal of consideration. Mackay has observed similar appearances in association with other pathological conditions and he expresses the opinion that the diminution in numbers of the platelets in *purpura hæmorrhagica* is also due to toxic action on the hæmopoietic system, that it is not due to excessive destruction of platelets and that it is in no way concerned in the ætiology of the disease. It is interesting to note that splenectomy is followed by an increase in the numbers of platelets; Mackay takes this as evidence that the spleen is concerned in the destruction of platelets.

Numerous observers have suggested that platelets are of importance in the coagulation of blood.

[1] *The Quarterly Journal of Medicine*, April, 1931.

Many have expressed the opinion that certain blood dyscrasias, such as hæmophilia, are due to abnormalities of the platelets. As a result of his investigations Mackay concludes that the platelets probably accelerate coagulation, but that it is doubtful if they are of more than secondary importance, and that a knowledge of the numbers of platelets in the peripheral blood is no guide to the rate at which clotting will occur after blood is shed. The numbers of blood platelets appear to have no influence on the occurrence of spontaneous capillary hæmorrhage nor on the length of bleeding time. The most important factor concerned in capillary bleeding is the ability or inability of the vessel walls to contract.

There is much more of interest in the paper, which should be read by all those especially interested. In addition to an account of Mackay's investigations, it contains a review of the literature.

Mackay has disposed of several difficulties in the way to a clear understanding of the part played by the blood platelet in human physiology and pathology, and he has done a service in arousing further interest in a constituent of the blood of which extremely little is certainly known. At the same time he has been able to give no hint as to the function of the platelet. Doubtless the limitations of the microscope have something to do with the present lamentable lack of knowledge concerning blood platelets. It is important that a great deal more investigation should be carried out, for the blood platelet is influenced by physiological changes and pathological conditions, and there can be little doubt that it is a cellular element with definite functions.

FILARIASIS.

DOUBTLESS few people will ever think of *Filaria bancrofti* by any other name, but it has been decreed that *Wuchereria* is the correct generic title; presumably it must be tolerated; at any rate, many worms have worse names.

Filarial infestation and filarial disease present numerous puzzling features. Acton and Sundar Rao in India, and Buxton in the Pacific Islands have done a great deal recently towards clearing up these difficulties, but much remains to be done. It has long been believed that elephantiasis is caused by infestation with *Wuchereria bancrofti*, but S. L. Brug's recent investigations reveal the probability that another worm, which he has labelled *Filaria malayi*, may often be the chief factor. The adult *Filaria malayi* has not yet been discovered, but the microfilaria was described by Brug in 1927. The mosquito *Culex fatigans* is the commonest, though not the only, vector of nocturnal *Microfilaria bancrofti*, but in 1927 Lichtenstein in Sumatra found that certain microfilariæ which appeared nocturnally in the peripheral blood of man did not develop in *Culex fatigans*; Brug discovered differences between these microfilariæ and *Microfilaria bancrofti*. Since that date there has been a good deal of further work, which Brug discussed briefly before the Section of Tropical Diseases and Parasitology of the Royal Society of Medicine.[1]

The differences between *Microfilaria bancrofti* and *Microfilaria malayi*, according to Brug, are best seen in films stained with hæmatoxylin. The nuclei of *Microfilaria bancrofti* are arranged neatly and may be easily counted, those of *Microfilaria malayi* are more clotted together and are counted with difficulty; the tail of *Microfilaria bancrofti* is devoid of nuclei, that of *malayi* possesses one, two or three nuclei, one of which is situated at the end. The anal pore of *Microfilaria malayi* is clear and readily distinguishable, that of *Microfilaria bancrofti* is ill-defined and may be indistinguishable. Brug at first thought that *Microfilaria malayi* may have represented a stage in the development of *Microfilaria bancrofti*, but on maturer consideration he realized that other investigators would have observed the caudal nuclei if this were indeed a developmental form, also *Microfilaria bancrofti* may be entirely absent in regions where *Microfilaria malayi* is abundant and *vice versa*. De Rook and Brug in 1930 concluded that four species of mosquitoes belonging to the genus *Tæniorhynchus*, subgenus *Mansonioides*, are capable of transmitting *Microfilaria malayi*. Members of this subgenus are common in parts of Australia. In the mosquito there are certain appearances of the developing larva which further distinguish it from *Microfilaria bancrofti*.

The most interesting and important feature of Brug's paper is his conclusion that the adult *Filaria malayi* may cause elephantiasis. In certain areas in the Dutch East Indies *Microfilaria malayi* is common, while *Microfilaria bancrofti* is absent. In such areas elephantiasis of the limbs, especially the legs, is frequently seen, while other obvious manifestations of filariasis are rare. Further investigation is necessary before it can be said whether such conditions as chyluria, lymph scrotum *et cetera* may be caused by infestation with *Filaria malayi*. If Acton's conclusions with regard to the causation of these various manifestations of filariasis be correct, it may be reasonable to assume that chyluria *et cetera* may be produced by *Filaria malayi* and that the rarity of filarial diseases other than elephantiasis in the Dutch East Indies may be due to the great prevalence of the insect vector and consequent gross infestation. This feature was discussed in these pages in the issue of June 28, 1930.

The importance of *Filaria malayi* is not merely academic. There is a possibility that its incidence may be widespread and that further endemiological studies and studies of the adult worm, when it is found, may reveal much valuable information concerning filarial disease. Of course, until the adult worm is found and described, *Filaria malayi* must remain a hypothetical creature; the doubter will remark that *Microfilaria malayi* may be but a variety of *Microfilaria bancrofti*, and his suggestion cannot be entirely disregarded.

[1] *Proceedings of the Royal Society of Medicine*, April, 1931.

Abstracts from Current Medical Literature.

SURGERY.

Spinal Anæsthesia in Abdominal Surgery.

L. S. McKITTRICK, W. L. McCLURE AND R. H. SWEET (*Surgery, Gynecology and Obstetrics*, April, 1931) discuss the value of spinal anæsthesia in abdominal operations. Satisfactory anæsthesia for a surgical operation within the abdomen requires a loss of the sensation of pain and sufficient relaxation of the muscles to permit the surgeon to perform the operation without loss of time and with a minimum of trauma. Spinal anæsthesia does not fulfil all of the requirements essential to the ideal anæsthetic for abdominal surgery. Nevertheless, it has an important place in surgical operations below the diaphragm. The indications for and the contraindications to its use are difficult to enumerate. It is safe in experienced hands. It is indicated in operations lasting an hour to an hour and fifteen minutes, in the presence of diabetes, tuberculosis or of definite damage to the kidneys or liver. It is indicated in long operations whenever it is desirable to spare the patient an hour or more of general anæsthesia. It is contraindicated in perforations of the intestines because, through its effects on peristalsis, intestinal contents may be emptied into the abdominal cavity. It is also contraindicated in treatment of a patient on the verge of collapse, when the nature of the condition, such as general peritonitis, forbids the use of the Trendelenburg position, whenever a sudden fall of blood pressure might be hazardous, and for any operation on a very sick patient which can be successfully carried out under local anæsthesia. There is no evidence that hypertension *per se* is a contraindication nor increases the risk. The advantages of spinal anæsthesia are complete relaxation, quiet respiration, contracted intestines, a probable lack of direct irritation to kidneys, liver and lungs, and the absence of any direct effect upon the metabolism of a patient with *diabetes mellitus*. The disadvantages are the short, uncertain period of anæsthesia, the uncertainty of its effect upon blood pressure, occasional nausea and vomiting occurring during operation and the slight handicap of working in the upper part of the abdomen with the patient in the Trendelenburg position.

Fibrous Peritonitis of Neck of Hernial Sac.

V. L. SCHRAGER AND J. T. GAULT (*Surgery, Gynecology and Obstetrics*, April, 1931) describe fibrous peritonitis of the neck of the hernial sac. Fibrosis within the hernial sac is quite a common occurrence. The authors are concerned, however, only with the circumscribed ring-like fibrosis occurring at the neck of the sac, which has a definite anatomical and pathological significance. The ring has been found by the authors to be always flush with the internal ring, it is very tough, and the rest of the surface of the sac has been found perfectly smooth and apparently quite normal. It has been found in indirect but not in direct hernia. Diffuse, inflammatory changes within hernial sacs are fairly well known and recorded. Peritonitis of hernial sacs, according to Piquet, is more common in umbilical than in inguinal herniæ. Fibrous peritonitis at the neck of a hernial sac is a rather common occurrence. This circular fibrous ring at the neck of the sac is very likely the result of an aseptic traumatic peritonitis due to repeated muscular contractions at the internal ring. Fibrous peritonitis is responsible for a number of strangulations, the degree of damage being proportionate to the toughness of the ring and the narrowness of the lumen at the neck of the sac. Reposition of viscera in severe strangulations frequently requires preliminary division of the fibrous ring, both as an aid in replacement and as a safeguard against mechanical damage to the bowel wall and its mesentery. The authors are unable to identify the internal sphincter of MacGregor, which may be due either to inexpertness in dissection or insufficient anatomical material. The internal oblique and transverse muscles exert a "shutter-like," possibly a "sphincter-like," action, which satisfactorily opposes the intraabdominal pressure.

Prophylactic Treatment of Thrombosis by Thyroxin.

K. BOSHAMER (*Münchener Medizinische Wochenschrift*, March 6, 1931) discusses the use of thyroxin in preventing post-operative thrombosis. The success which at first attended this treatment has not been maintained, although it has reduced to some extent the frequency of the condition. The particular constitution which renders a patient liable to thrombosis is defined as being one in which the parasympathetic nervous system (vagus) dominates the sympathetic. This can frequently be detected by a slowing of the pulse rate after operation in spite of administration of thyroxin which usually accelerates it. The observation of the pulse-temperature curve is useful as giving an indication of a likelihood of a thrombosis. The patient whose pulse remains slow in spite of thyroxin treatment, is to be regarded as an extremely probable candidate. The author recommends the use of ephetonin for the latter type of patient.

Hare Lip and Cleft Palate.

ALBERT D. DAVIS (*Surgery, Gynecology and Obstetrics*, April, 1931) describes the surgical correction of cleft lip and palate. Operative failures are estimated at 70% to 80% of cases. Many prominent surgeons have abandoned palatoplastic surgery because of the preponderance of failures. In the light of present-day knowledge the proportionate number of failures is too high. The author makes a plea for a better understanding of the anatomy and functional activity of the palate. The fundamental value of the Brophy bone operation in early infancy cannot be overestimated. Failure of union results in abnormal bony arch, abnormal occlusion of teeth, short, tense, soft palate, and many failures of union, imperfect speech as a result of the short, tense palate, imperfect foundation for lip or soft palate operation, imperfect foundation for orthodontic treatment, with probable increase in cleft bones if movement is attempted. The importance of the time of operation and the sequence of operations cannot be too greatly stressed. The spreading of the tuberosities when single wire sutures are placed causes difficult approximation of the soft tissues, oftentimes necessitating the use of lateral incisions, which are condemned; it causes malocclusion of teeth; operations are also required to lengthen the palate in order that perfect speech (which is the goal of the soft tissue operation) may be obtained. Mere closure of the defect without regard to anatomical and functional result is a poor substitute. The cooperation of a pædiatrician has accounted for a decrease in mortality.

Mammary Cancer in Relation to Sexual Life of Cuban Women.

JUAN A. DEL REGATO (*Vida Nueva*, April, 1931) publishes the results of his investigation into the influence of race, age, civil state, sterility and lactation on the incidence of mammary cancer in 150 Cuban women. The married sufferers constituted 65% and the unmarried 35% of this number. Of the sufferers 83%, however, had led an active sexual life. He confirms Roffo's opinion that cancer of the breast is more frequent in women leading an active sexual life who do not bear children. It is suggested that the Japanese woman suffers less from cancer of the breast than the English woman, because it is a more common practice to suckle children in Japan. The author states that in 47% of his patients there had been irregularities in the function of lactation. Sixty *per centum* of the sufferers belonged to the white race and 29% were nulliparous.

The Ætiology of Thrombosis.

FELIX FRANKE (*Münchener Medizinische Wochenschrift*, March 6, 1931) puts forward the theory that the frequency of venous thrombosis at present observed in numerous centres is due to an infection and in particular to a chronic influenza. He quotes several cases in which thrombosis appeared as a definite symptom of an influenzal attack and is certain that the large number of inexplicable post-operative thromboses have really a latent influenzal basis. The pathology of the condition he defines as an inflammation of the vessel wall

coupled possibly with a diminution in the cardiac efficiency. Evidence of such changes is seen in the tendency during influenza to hæmorrhage of various kinds, from nose, gums, pharynx and larynx and also sometimes as menorrhagia, metrorrhagia and even as *purpura hæmorrhagica*. He is disinclined to attribute the thrombosis to a traumatic influence. By a consideration of a series of influenza epidemics he is able to show that at different times the chief symptom of influenza may vary, one epidemic being characterized by affection mainly of the lungs, another of the joints and nervous system. The present prominence of thrombosis, he asserts, is a temporary expression of the disease in the vascular system, which will probably soon disappear. He admits that prophylaxis is difficult, but has had success in the treatment of the thrombosis by administration of digitalis, sodium salicylate and antipyrin.

The Treatment of Empyema.

JOSEPH A. DANNA (*The Journal of the American Medical Association*, May 2, 1931) reports thirty-five patients with empyema who have been treated by him since 1923 by a method which he first saw used in Italy. He aspirates the purulent fluid with a large syringe. After each syringeful is extracted, a corresponding amount of air is introduced. He states that by so doing the volume of the lung is not rapidly altered and the position of the mediastinum remains practically unaltered. He has removed as much as 3,000 cubic centimetres at one sitting. The procedure is repeated as often as fluid reaccumulates, on an average every sixth day. When possible, the patient is kept in a sitting position during operation. He stresses the great rarity of respiratory or cardiac embarrassment with this method. He has been surprised at the small amount of pus which forms after aspiration, in contradistinction to the large amount which drains following a thoracotomy. Should masses of fibrin form during treatment, time should be allowed for these to liquefy or a small incision may be made under local anæsthesia and the masses driven out by forced respiration. He has tried this method in a case of prepatellar bursitis, one case of subphrenic abscess and one case of suppurative arthritis of the knee joint, in each with marked success. He suggests that the method be given a trial in purulent pericarditis and pyogenic infections of the cerebral meninges.

Duodenal Diverticula.

A. WINTEROLL (*Klinische Wochenschrift*, April 11, 1931) states that diverticula may occur in any portion of the duodenum, but are found most frequently near the ampulla of Vater. They may consist of a herniation of the mucosa through the muscular coats or else be a true diverticulum. They are generally multiple, possess a narrow neck and vary in size from a bean to a hen's egg. Clinically the first signs noted follow inflammatory changes produced by the pent-up contents of the sac. This may be associated with perforation and general peritonitis. Cholangitis and pancreatitis may complicate the picture because of the anatomical site of the diverticula. However, the majority cause few symptoms beyond indefinite pain in the upper part of the abdomen and are diagnosed only during routine radiographic examination of the intestinal tract. Pictures show a well filled shadow in the region of the duodenum. This persists after the duodenum is emptied and may be observed for several days. While the common site is in the descending portion of the duodenum, the inferior part and the duodeno-jejunal junction are also affected, but it is rare to find any diverticula in the first part. The majority of patients can be successfully treated along conservative lines—rest in bed and diet. The details of seven cases are given with radiograms of the lesions.

Surgical Treatment of Goitre.

J. BAUER (*Klinische Wochenschrift*, April 4, 1931) discusses the modern indications for the surgical treatment of goitre. He divides hyperthyreoidism into two groups—primary when the amount of gland substance involved and colloid formation are small, secondary when the colloid substance is greatly increased. The beneficial effect of preoperative Lugol's solution treatment is most pronounced in the first group. An absolute indication for surgical treatment is mechanical obstruction to the trachea which occurs with nodular growths and does not depend on the degree of hypersecretion. Operation is also indicated for all patients with severe goitre who have not improved or who have retrogressed after two or three months of medical treatment. Patients with goitres of moderate severity which fail to react to medical or X ray treatment after six months should also be submitted to operation. Such patients do not present the same dangers of hyperthyreoidism, and time can be spared to judge the effects of non-operative treatment. X ray treatment is indicated for the *forme fruste* types, for those who have had several operations without success, for elderly patients or those with severe cardiac lesions for whom operation is inadvisable. One of the main causes of failure after operation is insufficient removal of tissue, while in some it is due to hypertrophy of the thymus which can best be treated by resection.

P. KLOSE (*ibidem*) discusses recent advances in the preoperative and operative treatment of goitre. A period of rest in bed for one to two weeks is most essential, although at first many patients rebel at the delay. During this period the administration of Lugol's solution is most important—five to fifteen drops twice daily. He considers that radiation of the thymus is also useful. The dose is 60% of the minimum skin dose, and can be repeated in a fortnight. He does not consider that estimation of the basal metabolic rate is of much practical importance. The best time for operation depends mainly on the improvement noted after the above preoperative treatment. If this occurs rapidly, operation can be done within six to ten days; when improvement occurs more slowly, three weeks should be allowed to elapse before operation. It is important not to operate if the patient shows no improvement. A great advance in treatment has been the use of "Avertin" anæsthesia, with its prolonged effects after the actual operation. As a result of such preliminary preparation and the use of "Avertin" the author prefers to operate in one stage with ligature of the four arteries and subtotal resection of both lobes.

The Colon.

JOHN A. LICHTY (*The Journal of the American Medical Association*, February 28, 1931) considers that even after so-called chronic inflamed appendices and thickened gall bladders have been removed, many patients still suffer from chronic irregularities. He describes a syndrome consisting of a mild degree of invalidism with psychic and neurotic symptoms, irregularity of the bowels and annoying borborygmus, soreness along the course of the colon with an occasional pain and bearing-down sensation in the lower part of the abdomen with gradually developed food phobia, leading to loss of weight. The diagnosis may be definite, but the examination must in all cases be thorough and should include a barium enema. In the early stages of treatment it is wise to avoid much roughage and all irritating purgatives. Constipation will be obviated if sufficient fats and fluids are included in the dietary. Magnesium oxide and belladonna are useful when spasticity is present. If the bowel is atonic, small doses of cascara and *nux vomica* are indicated. It is a great mistake for these patients to form the habit of flushing the colon; this procedure only becomes a useless fetish. Apparently this practice dates back to the ancient Egyptians, who watched the ibis wash his beak and then apply it to his preen gland preparatory to preening his feathers. They were under the mistaken idea that he was submitting to lavage of the colon. It is stated that Louis XIV received several thousand intestinal douches in the course of his life, and the renowned Duchess of Alva treated her dying son with enemas as an "emulsion of religious relics." Recently researches have demonstrated the futility of the so-called high enema. Within a few minutes of the commencement of the administration of an ordinary enema the fluid reaches the caecum. Nothing can be gained by coiling several feet of rubber tubing in a dilated rectum. Atony is prone to follow the distension of the colon by quarts and even gallons of fluid.

Special Articles on Diagnosis.

(Contributed by Request.)

LV.

OBSTRUCTION OF THE SMALL BOWEL.[1]

As students and residents we become familiar with two grades of intestinal obstruction, the very acute and the very chronic. In the first, acute obstruction of the small bowel, the clinical picture is usually complete within twenty-four hours. In the second, obstruction of the large bowel, the clinical picture is not fully developed for months.

The object of this paper is to call attention to a group of cases which lies between these two grades. This is an obstruction of the small bowel, but it is an obstruction of such a nature that the symptoms and signs characterizing obstruction may not be apparent for two or three days, or, as in the first case referred to later, for weeks.

The syndrome of intestinal obstruction is made up of four main symptom groups: (i) Recurrent colic, (ii) vomiting, (iii) non-passage of fæces or wind, (iv) when the obstruction has been present for sufficient length of time, distension and visible peristalsis.

In the acute obstruction of the small bowel that is usually seen, the symptoms rise rapidly to a height and the first three of these symptom groups are apparent soon after the onset. The colic is severe and recurs at frequent intervals. Vomiting comes on soon after the onset and its continuance is a feature of the condition. Stoppage of the bowels is complete, although an enema may produce one good result. Distension, of course, means failure of the bowel action and, if it appears in this grade of obstruction, it is taken to mean that diagnosis has been too long delayed. Once distension does appear, it rapidly becomes generalized. The condition is so acute that it cries out for immediate attention. Diagnosis is urgently demanded and fortunately is not usually difficult.

The grade of obstruction about to be described differs from the very acute grade in that, although the symptom groups are identical, the condition is drawn out to an unusual degree, and there are long silent intervals between the appearance of the groups. One's sense of danger is not sufficiently aroused and diagnosis is often put off until it is too late. The first case to be reported is an example of how this may happen. It is not quite typical of the grade of obstruction to be described, as it is drawn out to a degree altogether unusual. Nevertheless it does bring out the marked differences between the two grades of small bowel obstruction.

CASE I: A man, aged forty, was admitted to hospital on a Wednesday afternoon. He stated that for three years he had been subject at intervals of months to attacks of moderately severe abdominal colic. The pain started in the left upper quadrant and worked down to the left loin. One month before admission he had an attack which was more than usually severe. It subsided in a few days, but since that time had at intervals of days recurred. He had not vomited. His bowels had opened daily. He had some frequency of micturition during the attacks. He had been told that he had a stone in the kidney, and he did not wish to be operated on.

On examination he was a sickish looking man. He indicated the pain as being vaguely to the left of the umbilicus when it appeared. He was not in pain at the moment. General examination revealed no abnormality. There was no rigidity of the abdomen. There was no tenderness. In the left upper quadrant there was a suggestion of distension. The remainder of his stay in hospital is summarized as follows.

On Thursday morning an X ray examination of the renal tract was made. The radiologist reported later in the day the presence of a "small opaque body in the line of the left ureter, possibly a ureteric calculus." At 1.30 p.m. the patient had an attack of acute abdominal pain. This was relieved by morphine. At 7 p.m. he had another attack of pain and vomited a receiver full of vomitus. The bowels were reported not to have opened.

On Friday at 2.30 a.m. the patient had a severe attack of pain. Less severe attacks occurred later. At 7 a.m. an enema was given with fair fæcal result and relief to the patient. At 11 a.m. an opaque ureteric catheter was passed on the left side and a photograph was taken. The radiologist reported later that the opaque body formerly seen was off the line of the ureter. At 4.30 p.m. the patient had an attack of pain, and at 6.45 p.m. he vomited.

On Saturday morning the distension to the left of the umbilicus was undoubted. The diagnosis of intestinal obstruction was made. It was thought that the obstruction was in the large bowel. The patient did not clearly understand the position and declined operation. On being pressed, he asked for twenty-four hours to consider it. This was foolishly granted. An enema was given with fair fæcal result and great relief. On the Saturday morning operation was performed. Distended coils of small intestine presented. The patient's condition went off and an ileostomy was performed and the abdomen closed. He died some hours afterwards.

The *post mortem* examination was surprising. There had been a recent acute appendicitis which had subsided, leaving the appendix lying in a small pool of pus. The appendix was to the left of the mid-line, at the brim of the pelvis, and the abscess was between it and the posterior wall. The sigmoid was adherent to the inflammatory mass forming a band. Beneath this band ran the last few inches of the ileum. This was the site of obstruction, but the obstruction was only partial. It is possible that the attacks of pain he had had for years were recurrent attacks of appendiceal colic. The acute attack had been recent, but not very recent. The most probable time to place it would be coincident with the acute pain he had one month prior to admission. The attack subsided, but resulted in the obstruction described. The obstruction was only partial and the patient's bowels acted daily. From time to time, however, the small intestine made attempts to throw off the obstruction. Eventually the partial obstruction was converted to a complete or nearly complete obstruction by the failure of the bowel close to the obstruction.

This failure became apparent as distension of the lower part of the bowel, and because of the position of the obstruction and of the disposition of the intestinal coils this was at first localized and seen to the left of the umbilicus.

This case, of course, is a very unusual one, but it does bring out, although in an exaggerated way, the distinctions between this syndrome and the syndrome of very acute obstruction.

These differences are: (i) The long intervals between the attacks of colic. This is the outstanding feature and is a complete contrast to the rapid succession of attacks that is characteristic of very acute obstruction. (ii) The fact that vomiting may not occur till there have been many attacks of colic. (iii) That enemata may produce fair fæcal results and may give great relief to the patient. This same point has been brought out in a recent article by J. Colvin Storey.[a] (iv) What is most important, that the distension is at first slight and localized, progressing only very slowly to the generalized distension seen in very acute obstruction. This localized distension is the outstanding physical sign and if X ray examination is not available, diagnosis may not be possible until this sign has appeared. Distension, of course, means failure of the bowel, but if the distension is local the failure is local and there is still time to act. Once distension has appeared, however, action must be immediate. In the case just described we entirely missed the significance of the localized distension.

CASE II: A man, aged twenty-six, had an "interval" appendix removed through a McBurney's incision. His bowels were opened with castor oil on the second day. All went well until the fifth day, when he complained of an

[1] This paper was read at a meeting of the Northern Division of the Tasmanian Branch of the British Medical Association on April 23, 1931. Dr. Craig has kindly consented to its inclusion in this series of special articles.

acute attack of colic round the umbilicus, which lasted for half an hour. His bowels had opened that morning. Events may be summarized thus:

On the fifth day, at 3 p.m., the patient had a first attack of colic, but was quite well after it.

On the sixth day, at 2 a.m., he had another attack of colic lasting for the same time, but slept well after it. At 10 a.m. he had another attack. An enema was given with good result. The patient was relieved by it. Soon afterwards he was smoking and reading, and his abdomen was quite flaccid. At 11 p.m. he had another attack accompanied for the first time by vomiting. The abdomen was as before.

He slept well till the seventh day at 7 a.m., when he had another attack, again accompanied by vomiting. He was given another enema, which produced a fair fæcal result and again gave relief to the patient. At 9.30 a.m. he was found apparently quite well and arguing with his neighbours. Examination revealed a slight but unmistakable distension in the middle part of his abdomen.

At 11 a.m. operation was performed under ethylene and oxygen anæsthesia. The bowel was found acutely kinked by an adhesion to the parietal peritoneum in the region of the previous operation. This was relieved and he recovered well.

This case is a typical one and brings out well the features of the syndrome—the long intervals between the attacks, the fact that vomiting may not occur till there have been many attacks of pain, the fact that an enema may produce a fæcal result, the syndrome being completed on the second day by the appearance of localized distension. Further, it brings out the fact that after an attack of colic has passed off, the patient may be found in excellent condition reading the paper, smoking or arguing.

CASE III: A man, aged sixty-eight, stated that three days previously he had had an acute attack of abdominal pain round the umbilicus. Since that time it had been repeated at intervals of a few hours. After the first few attacks he had vomited with each pain. His bowels had not opened, nor had he passed flatus. Examination revealed a man in very good condition, cheerful, and with a clean tongue and slow, steady pulse. Examination of the abdomen, however, revealed a slight but undoubted distension, mainly in the right lower quadrant.

Immediate operation was ordered. When the peritoneum was opened, distended small gut presented. As no lump could be felt and non-distended small bowel could not readily be seen, the distended bowel was followed along. After several feet had been traversed, a spot was reached where there was a transition from distended to non-distended coils. Across this spot was a strongly marked impress of a band. The gut had evidently run beneath a band which had been broken by manipulation. It was impossible to say what part of the bowel was obstructed, but the impression was that it was the lower. Ileostomy was performed and the abdomen closed. Recovery was uneventful.

An interesting feature of this case was that an alert and experienced resident medical officer, on reporting it, said: "The symptoms suggest intestinal obstruction. His condition is so good after three days, however, that I think he must be exaggerating his symptoms. Even if he is not, I think he will wait till the morning." I report this because it is exactly what I would have said myself, had I not had previous experience of such cases. The key to the diagnosis was the slight localized abdominal distension. A further point is that the condition of these patients may change rapidly, so that a patient who is in good condition in the evening, may in twelve hours' time be almost inoperable.

Explanation of the Symptoms.

The most likely explanation of the symptoms is that the obstruction is low down in the small bowel. *Post mortem* examination showed that in the first case the obstruction was at the very end of the ileum. In the other two cases the obstruction was most likely to have been low down. Another explanation is that the obstruction is incomplete, or, if complete, is at times partially relieved. In the first case the obstruction must have been only partial for some time. When obstruction is due to a kink it is understandable how the obstruction may be relieved should movements of the bowel partially flatten out this kink.

Diagnosis.

Before speaking of diagnosis in particular there are two points to be made in regard to diagnosis in general.

The first point is this: the great difficulty in diagnosis is that we know so little about the production of symptoms. Did we know how symptoms were produced, it might be possible in any given case to make a diagnosis from the contemplation of the symptoms alone, apart from any previous experience of such symptoms. This, however, we cannot do. Diagnosis depends almost solely on a vast previous experience.

Over a great number of years symptoms have been grouped and regrouped, analysed and reanalysed, until a great number of syndromes have been separated, each syndrome corresponding to some disease or phase of disease. Our method of diagnosis in any particular case is, by means of history taking, examination and special investigation, to build up a clinical picture. We then compare this clinical picture with the standard clinical pictures, making the diagnosis from this comparison.

This method has many advantages, but also many disadvantages. The great disadvantage, as every doctor knows who has a large out-patient department or general practice, is that the majority of patients complain of symptoms which belong to no known syndrome. Diagnosis is merely a conjecture in most of these cases and treatment is symptomatic. But further than this, even when a patient does appear with a well defined syndrome, it does not mean that diagnosis is certain. I always think that in the account of almost every disease there should be included under the heading of "differential diagnosis" a special paragraph for "similar symptoms of an unknown origin."

The cause of even such a well defined syndrome as acute abdominal colic may never be discovered. We have had in hospital recently patients with recurrent acute abdominal colic in regard to whose condition the utmost investigation failed to reveal a cause. It is this knowledge that in the majority of cases our utmost efforts will produce no result, that makes diagnosis so difficult. It subconsciously breeds in us a tendency to defer diagnosis, in the hope that, as so often happens, the symptoms will pass off without special treatment.

The second point is this. The hospital surgeon has an enormous advantage over the general practitioner in making a definite diagnosis, inasmuch as he rarely sees a patient until the clinical picture is complete. On the other hand, the general practitioner sees the patient first when there may be only one symptom present. The possibilities of development are many. Even as symptom after symptom is added to the picture the possibilities only narrow down slowly. It is, of course, very interesting to watch the development of a syndrome in this manner, but the whole time importunate relatives are waiting, anxious for a clean-cut diagnosis. For this reason the hospital surgeon will often get undeserved praise for making the diagnosis in half an hour, when the general practitioner was jumping from one thing to another for two days.

In working out a differential diagnosis, therefore, it is not of much value to recite the details of developed syndromes. We should commence, as the general practitioner has to commence, with the first symptom.

We will assume that he has been called at 8 a.m. to see a patient with acute abdominal pain. The first thing to do is to try to distinguish the type of pain. With a few rare exceptions the syndromes of acute abdominal conditions fall within four well marked classes—the syndrome of acute inflammation, the syndrome of perforation, the syndrome of colic, and the syndrome of acute torsion. The majority fall within the first three. Each class has its definite mode of onset and of progression. Fortunately, in the cases under discussion it is not difficult to distinguish them as due to colic. The patient will be found doubled up and holding his abdomen. His abdominal

wall will be held too rigidly for much to be done in the way of examination. After a while, however, the pain will relax a little and the patient will breathe more freely. His abdominal wall will move easily and will be soft and not tender. Soon, however, the pain will come again. It will continue to wax and wane in this way until finally it passes off. During the pain the patient may complain that it is "gripping him" or that it is "going round and round." If the pain can be distinguished as a colic, it must be due to violent peristaltic contracture of one of the hollow muscular tubes within the abdomen.

These tubes are: gall bladder and the bile ducts, the stomach and intestines (including the appendix), the pancreatic duct, the pelvis of the kidneys, ureters and bladder, the uterus and tubes. We are discussing a male patient, but with a female patient the gynæcological history and a pelvic examination done as soon as the pain has subsided, should put out contracture of the uterus or tubes as a cause of the pain. Colic of the pancreatic duct must be a great rarity.

This leaves the gall bladder and bile ducts, stomach, intestines and appendix and the renal tract. The thing to do now is to think out the possibilities. The possibilities are:

1. Colic of the gall bladder or bile ducts, usually due to stone, but occasionally to hydatid cysts. Gall bladder pain usually declares itself by its particular distribution, but often it does not do so.

2. Colic of the renal tract, due to stone, blood clot or oxalates. This pain again often, but not always, declares itself by its particular distribution. In my own experience renal tract pain is less likely to be typical than gall bladder pain.

3. Colic of the intestines, due to food poisoning, intestinal obstruction or to lead poisoning. The pain of intestinal colic is usually referred to the mid-line, but it may be felt towards one side or the other.

4. Colic of the appendix, due to the obstruction.

5. Colic of unknown origin.

It is quite possible that this colic may never return, or even if it does return, we may never discover its cause. We should, however, disregard this knowledge and proceed as if diagnosis were certain.

At this stage, when the patient has had one attack of colic which has passed off, diagnosis will be impossible. The outstanding question at the moment is whether or not the patient is suffering from food poisoning. There are, of course, varying degrees of food poisoning. If it is a really severe attack, the symptoms will not cease for more than a short time. Pain will be followed by vomiting and vomiting by diarrhœa. As a rule the patient knows that he has eaten bad food, and often others have eaten it too, and are also sick.

Lead poisoning may also be eliminated by an inquiry into its possibility and by examination for the blue line on the gums, paralyses *et cetera*. The urine should be examined microscopically, particularly for pus, blood or oxalates. Beyond this, all that can be done is to advise the patient to remain at rest and to promise to return in three or four hours, as by that time, if anything acute is going to happen, something further will be manifest. When the doctor returns in three or four hours' time, he will find the patient in good condition, in no pain and quite cheerful. He may then feel fairly certain that it is not a case of serious food poisoning, and further, that it is unlikely to be either acute apendicular obstruction or acute appendicitis, both of which conditions progress in the first few hours. This leaves biliary colic, renal colic, intestinal obstruction and colic of unknown origin. Finding the patient so well, however, the doctor will probably be content to leave the matter at that for the moment.

At 9 p.m., however, he is called again to the patient, who is suffering from another attack of colic exactly similar to the first. The physical signs are as before. Knowing by experience that the reference of pain is often not true to type, the physician is still uncertain as to whether the pain is due to colic of the biliary tract, renal tract or intestines. He feels now that he must have at once some further information. The urine should again be examined. Whether anything or nothing is found, the renal tract should now be examined by X rays. The fact that it is an awkward hour at night should not prevent this. Though a small stone may escape, a stone of any moderate size will be seen. If there is any doubt as to whether it is in the renal tract an opaque catheter should be passed at once and another photograph taken. If no stone is seen and nothing abnormal is found in the urine, the diagnosis is narrowed down to biliary colic, intestinal obstruction and colic of unknown origin. An enema should now be given. Unless the patient opened his bowels shortly before the first attack, the result will be good. The physician now knows that the lower part of the bowel is empty.

The next thing to do is to give a barium meal. So far we have not used this method of diagnosis in these cases, but it seems the obvious thing to do, and one that, theoretically at least, must give a diagnosis. A barium meal reaches the caecum in six hours. If, then, in eight hours' time most of the meal is in the colon, the condition is obviously not one of small bowel obstruction. If, on the other hand, there is obstruction, only a small part or none of the meal will have passed a certain spot. By screening again, say, an hour later, it will be seen that the bulk of the meal is still held up. Moreover, the barium should show up the early dilatation of the small bowel before it becomes obvious to external examination.

If the physician is so situated that a barium meal is impossible, there are two physical signs to rely on—the results of enemata and the appearance of localized distension. As has been shown in the reports of the cases, the results of enemata may be fallacious inasmuch as moderately good fæcal results are often obtained and great relief is given temporarily to the patient. If this is so, the only thing to do is to make repeated examinations for slight localized distension, which may appear in any quadrant of the abdomen, according to the disposition of the small bowel.

It may be said that in cases of recurrent abdominal colic in which there is a localized distension which is not immediately and completely relieved by enemata, immediate operation is justifiable.

Reference.

(1) J. Colvin Storey: "Obstruction of the Small Intestine," THE MEDICAL JOURNAL OF AUSTRALIA, May 3, 1930, page 586.

C. CRAIG, M.D., M.S. (Melb.), F.R.A.C.S.,
Surgeon-Superintendent, General Hospital, Launceston.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE WESTERN AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Perth Hospital on May 20, 1931, DR. H. J. GRAY, the President, in the chair.

Common Ocular Conditions.

DR. D. G. GAWLER read a paper entitled: "Common Ocular Conditions in General Practice" (see page 121).

DR. D. D. PATON expressed appreciation of the paper and the manner in which Dr. Gawler had covered the field of ophthalmology of interest to the general practitioner. He considered that many general practitioners were very careful, while others had not the same judgement in deciding when a patient should be passed on. He sympathized with the man in the country, but considered that any patient with diminishing vision should be referred to the specialist. He considered that many patients with foreign bodies were referred too late, when infection of the injured site had taken place and active

ulceration was in progress. The general practitioner should never make a positive diagnosis of cataract unless using an ophthalmoscope.

He warned medical men against too energetic treatment of gonococcal ophthalmia. In these cases the corneal epithelium was softened and douching with too great a pressure resulted in damage to the epithelium with subsequent ulceration.

A subcutaneous injection of sterile milk, 0·5 to 2·0 cubic centimetres, was invaluable in these cases. The proprietary preparation "Aolan" could also be used, but milk was always accessible.

Trauma to the sound eye must be avoided in interstitial keratitis, as trauma undoubtedly resulted in the manifestation of the disease in the previously healthy eye.

Dr. C. MORLET also congratulated Dr. Gawler on his interesting presentation. He considered the condition of pterygium and cataract were often confused by patients, and pointed out the importance of operation in the former condition, before the pupil had been encroached upon and vision impaired.

Episcleritis was often confused with conjunctivitis. It was characterized by exacerbations alternating with periods of quiescence, its resistance to local treatment, and cure on elimination of toxic foci.

DR. B. HUNT stated that one ophthalmological condition dealt with by Dr. Gawler was of extreme interest to the physician, the condition termed renal retinitis. In view of the fact that the majority of the changes described by Dr. Gawler as making up the picture of renal retinitis occurred also in purely arteriosclerotic retinitis, Dr. Hunt asked Dr. Gawler whether there were any specific points of value in differentiating the two conditions in view of the extremely grave prognostic significance usually attributed to the discovery of renal retinitis.

He also asked Dr. Gawler why many patients with renal retinitis complained of transient diminution of visual acuity, occurring usually in the morning.

Dr. Gawler in reply acknowledged Dr. Hunt's difficulty, stating that the various types of renal retinitis merged imperceptibly one with another, but that he considered cotton wool patches to be a manifestation of nephritis alone and not present in arteriosclerotic retinitis. He thought that transient dimness of vision in such cases was a vascular manifestation probably due to temporary arterial spasm.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Prince Alfred Hospital, Camperdown, on May 14, 1931. The meeting tok the form of a series of demonstrations by the members of the honorary staff.

Spontaneous Pneumothorax.

DR. COTTER HARVEY showed three patients who were suffering from spontaneous pneumothorax.

The first was a butcher, aged twenty-one years. At the age of six weeks a sister sat on his chest and "dinted it" on the right side; it has remained flat ever since.

The patient was perfectly healthy as a boy and played football for his town till two years ago. He was treated for right-sided pleurisy three years ago and the chest was strapped for four days. He ceased playing football as a result of a heavy tackle which gave rise to shortness of breath. He made no attempt to play again, though he felt quite well. Twelve months ago a friend noted he was short of breath on talking, and advised him to see a doctor. His medical attendant diagnosed pneumothorax. He has kept perfectly well during the past twelve months, gaining weight and working hard. He came to Sydney to see if anything could be done about it.

On examination he was seen to be a healthy and muscular youth. The right side of the chest was flattened, with signs of pneumothorax without fluid. The heart was in the left axilla. This was confirmed by a skiagram. A reading was taken of the intrapleural pressure; it was +14, +20, suggesting valvular pneumothorax. Two thousand cubic centimetres of gas were removed and the pressure was −14, −4. The patient had a slight pain in the right side and a dry cough. He was quite well next day, the reading being −20, +14. Seven hundred and fifty cubic centimetres of gas were removed and the pressure readings were −14, −4. A skiagram was taken, but showed no change. The heart and mediastinum were fixed in position. Lipiodol was injected, showing some aerating lung tissue in the right lung, but no sign of pneumothorax in the pleura.

Dr. Harvey said that the points for discussion were the cause of the condition, how long it had been present, what treatment, if any, should be carried out, and the prognosis from the respiratory and from the cardiac points of view. He thought that there was probably a small valvular pneumothorax present which was open at low pressures, but which was sealed at a pressure of about +15 centimetres of water. This must be constantly operating, otherwise pneumothorax would be at atmospheric pressure and would eventually be absorbed.

Dr. Harvey's second patient was a cleaner, aged thirty-two years, who was first seen on August 20, 1930. He had four years' war service, was gassed and severe facial burns resulted.

He was treated for right-sided pleurisy twelve months and again six months previously. During the last nine months he had noted great fatigue, but no other symptoms. On August 16, 1930, he was cleaning a window on top of a ladder when he felt a sudden pain in his right shoulder and was unable to use his arm. He felt ill and very short of breath, and went home to bed. On August 19, 1930, a skiagram confirmed the diagnosis of spontaneous pneumothorax. He was feeling much better one week later. The sputum was examined for tubercle bacilli, but none were found. The patient applied for and was given workers' compensation for six months. During this period he remained well, with no further sputum and steadily gaining weight.

Dr. Harvey pointed out that the lung had been kept collapsed since by artificial pneumothorax, with refills, at intervals of two to three weeks, of 900 cubic centimetres of air. Ten weeks before the meeting the patient resumed his heavy work as a cleaner and had kept well, continuing his refills.

Dr. Harvey said that the condition was almost certainly due to tuberculosis, though no pulmonary infiltration could be seen on X ray examination. He drew attention to the absence of any effusion or evidence of active pulmonary tuberculosis, which was characteristic of this type of pneumothorax occurring in young people "out of a clear sky." The other points for consideration were the advisability of keeping the lung collapsed for some years, and the giving of compensation.

Dr. Harvey explained that the tear in the pleura had occurred at the site of an adhesion near the apex of the right lung, which was presumably the sequel of the past pleurisies.

Dr. Harvey's third patient was a lorry driver, aged thirty-one years, who was first seen on January 5, 1931. He gave a history of no previous illness or ill health. Seven weeks before being seen, while lifting bags of flour, he felt sudden severe pain in the right side, with shortness of breath. X ray examination revealed complete pneumothorax. He spent two weeks in bed and five weeks off work, during which time the lung reexpanded. He returned to work feeling quite well and continued for two weeks on light work. Then he lifted bags of cement for two days. During the second day he noticed a return of pain in the right side and shortness of breath and had to stop work again. Pain ceased after two days, but dyspnœa persisted on any exertion and the patient has not worked since.

The patient was seen two weeks later with signs of right-sided pneumothorax, confirmed by screening (with no fluid). The left lung was clear. He was recommended for and obtained compensation, which he drew for two months. He was not seen again till two weeks before the meeting, having been at work for eight weeks. Dr. Harvey said that the patient kept fairly well, though he had cough

and sputum. No tubercle bacilli were found in the sputum and no evidence of pneumothorax was to be seen on X ray examination. No abnormal signs were to be found in the chest.

Dr. Harvey explained that this condition was similar to that of his second patient. Recurrent pneumothorax was a rare lesion and Dr. Harvey raised the question as to whether this patient should be given artificial pneumothorax. He pointed out that in the last two cases there was an absence of cardiac displacement, text books notwithstanding, and that in each the pneumothorax was closed and no fluid appeared.

Intermittent Hydrarthrosis.

DR. A. S. WALKER showed a married woman, aged thirty-one years. She complained of swelling of the right knee at intervals for the last four years. Eleven years ago the left knee became swollen painlessly and subsided in four to five days. This recurred frequently till four years ago. Then this ceased and the same process began in the right knee. During each pregnancy (the patient had two children) and period of lactation there were no swellings. The knee swelled rapidly. She could tell when a swelling was about to start and the fluid collected, reaching a maximum in a few hours, causing discomfort and a feeling of strain and weakness in the joint. It did not depend on injury and occurred during rest in bed for other causes. She had much local treatment to the joints.

Examination revealed no definite physical anomaly, save a certain degree of general asthenia. The Wassermann test gave no reaction. Skin sensitiveness tests were carried out in the Allergy Clinic, but yielded no reaction. Dr. Walker said that the joint during an attack felt slightly hot, but was not tender. It was tense, and after subsidence felt slack in the ligamentous structures. Fluid aspirated from the knee was clear, slightly viscous, contained no organisms on culture, but some neutrophile leucocytes. Swellings had recurred every few weeks, but were becoming less prolonged and frequent.

Among the forms of treatment tried were aspiration and strapping of the joint, with reinjection of ten cubic centimetres of effusion into the surrounding tissues; intramuscular injections of "Aolan," sterile milk and the patient's own whole blood; thyreoid and ovarian extract; iron and arsenic and dilute hydrochloric acid.

Cerebral Thrombosis Illustrating Apraxia.

Dr. Walker also showed a male patient, aged forty-four years, married, with three children. He complained of difficulty in speech. He had a "stroke" nine months ago, with sudden onset, but without loss of consciousness. The right arm and face were paralysed, but speech was not entirely lost.

On examination movements were fairly good in the right arm and face, though there was slight residual paralysis. There were definite signs of a moderate general arteriosclerosis. The systolic blood pressure was 170 and the diastolic pressure 110 millimetres of mercury. The serum did not react to the Wassermann test. The urine showed no abnormality. There was a definite grade of motor aphasia, but in addition this man illustrated how motor aphasia may be a variety of and associated with an apraxia, that is, an ability to perform certain movements of the purposive kind, even though the degree of movement and coordination he possessed were sufficient. Thus his right hand could not carry out acts like making use of common articles, such as a pencil or knife. When first seen he could not put his hat on with his right hand, though his arm and hand muscles could carry out all the necessary movements. These acts were performed without difficulty with the left hand. Dr. Walker said that probably the defect was not purely motor, but also in some degree ideational, due to some cortical or subcortical sensory loss. In spite of the fact that there must have been a lesion of definite magnitude in the distribution of the Sylvian artery he had improved, and practice and even repeated demonstration to students had effected some reeducation.

Still's Disease.

DR. KEMPSON MADDOX showed a boy, aged six years, who last year had an attack thought to be "rheumatic fever." Recovery was incomplete, leaving wasted limbs, painful, stiff and swollen wrists, elbows and knees. The child was irritable, anorexic, sweating and crippled. Very large tonsils and adenoids were removed, after which fever and sweating disappeared, with the exception of one exacerbation lasting forty-eight hours. Cervical adenitis gradually decreased, appetite, placidity and colour had improved.

Dr. Maddox drew attention to the characteristic attitude and expression, enlargement and synovial thickening of wrist, elbow and knee joints and of axillary and cervical glands (including posterior groups), absence of recognizable cardiac involvement or splenomegaly.

Dr. Maddox discussed the treatment of the condition and said that the prognosis was better than was at one time thought. Still's disease was better known as rheumatoid arthritis of children, since splenomegaly was not constantly present, nor did generalized adenopathy always occur. The epitrochlear glands were constantly enlarged.

Hæmophilia.

Dr. Maddox also showed a male patient, aged twenty-one years, who was suffering from hæmophilia. He had had "arthritis" since the age of seven years. He had always known himself to be a bleeder. He bled for two weeks recently after extraction of a single tooth. Joint effusions came on suddenly without warning or excuse; he felt sudden acute pain and noticed an immediate swelling which remained for two or three weeks, gradually becoming smaller and less painful, and associated with the appearance of altered blood beneath the skin surrounding the joint. The right knee joint was most frequently affected. No bleeding occurred into the alimentary, respiratory or urinary passages, but large deep bruises were constantly appearing in various situations for which he was at a loss for an explanation.

On examination anæmia was to be noted with oral sepsis, purpuric blotches, swelling, tenderness and limitation of movement in the right knee joint and right shoulder.

The capillary tube method of estimating the coagulation time revealed no clotting after thirty-five minutes. The bleeding time was fifteen minutes. The platelets were normal. A severe secondary anæmia was present. Occasional red blood cells were found in the urine. The blood calcium content was normal. Aspiration of the shoulder joint yielded blood.

The family history was quite free from bleeders among brothers or mother's ancestors. One brother examined had a normal clotting time.

Dr. Maddox said that he proposed to treat the patient with repeated small transfusions *plus* "Fibrogen" or "Thromboplastin" and intravenous injections of calcium, in order to shorten coagulation time sufficiently to allow of gradual eradication of the oral sepsis. Iron, arsenic, liver would be given in large doses.

In Dr. Maddox's opinion this was apparently a genuine case of sporadic hæmophilia with joint involvement. A family history probably existed, but could not be traced.

Pituitary Adenoma.

Dr. Maddox also showed a girl, aged sixteen years, who was suffering from an adenoma of the pituitary gland. The patient had been dull and sleepy for two months with progressive failure of vision and gain in weight. Dr. Maddox drew attention to the patient's mental condition, the acromegalic facies and hands, the increase in weight, the paresis of the left internal rectus muscle of the eye, and said that X ray examination revealed a somewhat enlarged *sella turcica*. He also stated that immediate operation would be undertaken by the transnasal route.

(To be continued.)

NOTICE.

THE Medical Secretary of the New South Wales Branch of the British Medical Association has forwarded for publication the accompanying list of books that have been added to the library of the Branch.

"The Soya Bean and the New Soya Flour," C. J. Ferrée; "Testicular Grafting from Ape to Man," G. Alexandrescu; "The Mycoses of the Spleen," A. G. Gibson; "Urinary Surgery," W. K. Irwin; "Practical Medicine Series": "Dermatology and Syphilis," "Urology," Series 1928, Year Book Publishers; "Percussion of the Chest," J. B. McDougall; "Diet and Care of the Surgical Case," R. H. Boyd; "Mechano-Therapy," M. R. Mulliner; "Colour and Cancer," C. E. Iredell; "Physio-Therapy in General Practice," E. B. Clayton; "Technique and Results of Skin Grafting," H. K. Christie; "The Basis of Epilepsy," E. A. Tracy; "An Introduction to Physical Anthropology," E. P. Stibbe; "A Synopsis of Medicine," H. L. Tidy; "Legal Medicine and Toxicology," R. W. Webster; "Hypertension and Nephritis," A. M. Fishberg; "Diet in Disease," G. A. Harropp; "The Sack-'em-up Men," J. M. Ball; "A Text Book of the Surgical Dyspepsias," A. J. Walton; "Abdomino-Pelvic Diagnosis in Women," A. J. Walscheid; "The Individual Delinquent," William Healy; "Neurosyphilis: Systematic Diagnosis and Treatment, presented in 137 Case Histories," E. E. Southard and H. C. Solomon; "Medical and Surgical Therapy," edited by Sir A. Keogh, five volumes, 1918; "Urgent Surgery," Felix Lejars, 8th French Edition, translated by Dickie and Ward, 1923; "Diseases of the Thyroid Gland," James Berry; "Albert's Diagnosis of Surgical Diseases," R. T. Frank; "Applied Anatomy, A Treatise," Edward H. Taylor; "Official History of the Australian Army Medical Services, 1914-18," Volume I: Part I, "The Gallipoli Campaign," Colonel A. G. Butler, D.S.O., V.D.; Part II, "The Campaign in Sinai and Palestine," Colonel R. M. Downes, C.M.G., V.D.; Part III, "The Occupation of German New Guinea," Colonel F. A. Maguire, D.S.O., V.D., and Captain R. W. Cilento, M.D.

A MEETING of the Sections of Neurology and Psychiatry, Oto-Rhino-Laryngology, Hygiene and Preventive Medicine of the New South Wales Branch of the British Medical Association will be held at the British Medical Association House, 135, Macquarie Street, Sydney, on August 14, 1931, at 8 o'clock p.m. The subject will be the "Effect of Noise on the Nervous System."

Dr. J. S. Purdy will speak on "Noise, a Nuisance Injurious to Health." Dr. Charles Warren will read a paper on "The Effect of Noise on the Ear." Professor Harvey Sutton will contribute to the discussion. Dr. C. Badham will speak on the effect of noise in industry. Dr. Donald Fraser will speak for the Section of Neurology and Psychiatry on the psychological effects of noise.

THE JACKSON LECTURE.

IN order to put on permanent record its appreciation of the distinguished services to the Branch of Dr. E. Sandford Jackson over a very extended period of time, the Council of the Queensland Branch of the British Medical Association has decided to establish an annual lecture in medical history, to be known as the "Jackson Lecture."

The first lecture will be delivered by Dr. Jackson himself on Friday, September 4, 1931, at the B.M.A. Building, Brisbane, at 8.15 p.m.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Grant, Alan Mostyn Bradford, M.B., B.S., 1929 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Dittmer, Felix Cyril Sigismund, M.B., B.S., 1930 (Univ. Sydney), Sydney Hospital, Sydney.

Smithers, Ernest Eric, L.R.C.P. and S. (Edinburgh), L.R.F.P. and S. (Glasgow), 1931, 3, Myreepi Flats, Ramsgate Avenue, Bondi.

Kenny, Rawdon Hamilton, M.B., B.S., 1928 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Medical Prizes.

THE CHALMERS MEDAL.

THE Chalmers Medal for 1931 has been awarded to Dr. N. Hamilton Fairley. The Chalmers Medal is awarded every second year by the Council of the Royal Society of Tropical Medicine and Hygiene in recognition of "research of outstanding merit" contributing to knowledge of tropical medicine or tropical hygiene. Dr. Fairley is at present a member of the honorary staff of the Hospital for Tropical Diseases, London. He is also Director of the Department of Clinical Pathology at the same hospital.

Obituary.

FREDERICK CHARLES STEVENSON.

WE regret to announce the death of Dr. Frederick Charles Stevenson, which occurred at Turramurra, New South Wales, on July 24, 1931.

EVAN TUDOR-JONES.

WE regret to announce the death of Dr. Evan Tudor-Jones, which occurred on July 26, 1931, at Sydney, New South Wales.

Correspondence.

POLYCYSTIC DISEASE OF THE KIDNEYS.

SIR: The following coincidence may be worth recording.

Two brothers, aged respectively twenty-six and thirty-four years, were in a truck which collided with a train. One was killed instantly. The other died twelve hours after admission to the Perth Hospital. *Post mortem* both were found to have polycystic kidneys. Each kidney of the younger brother weighed eleven ounces and showed several complete tears across it. The kidneys of the elder brother weighed forty-seven and fifty-three ounces. The larger kidney was intact, while the smaller one had split into two almost equal parts. In neither case were the ureters dilated nor was hæmaturia present before or after death.

Yours, etc.,

B. O. BRADSHAW.

Perth Hospital,
July 9, 1931.

COMMONWEALTH MEDICAL REFEREES.

SIR: One of the results of the present deplorable financial condition of the Commonwealth Government has been a very drastic change in the relations of the Old Age and Pensions Department and the Commonwealth Medical Referees and the work allotted to the latter.

I understand that in the capital cities at least the greater part of this work is now being done by medical officers of the Commonwealth Health Department, with consequent very grave reduction of fees to those men who for years

have served the Commonwealth well and faithfully in a work that, to say the least, has been not of the easiest.

With a view of organizing a protest against the action of the Government in this matter and of ascertaining the views of those medical men who have been acting as Commonwealth Medical Referees, I am in this communication requesting all of these gentlemen to send me their names and addresses together with any comments upon the present situation that they may consider of value.

The present situation can be remedied only by concerted action of some kind and I personally only wish to arouse to activity and coordination a group of men that are widely scattered over the whole continent.

Sydney would probably be the most appropriate headquarters for any such action.

Yours, etc.,

PAUL G. DANE.

110, Collins Street,
Melbourne,
July 20, 1931.

THE HIRE OF RADIUM.

SOME time ago the Senate of the University of Sydney purchased about 500 milligrammes of radium for use in the treatment of persons suffering from cancer. This radium is dispensed in ten plates containing from four to thirty milligrammes each, in ten tubes, each containing five milligrammes, and ten tubes each containing ten milligrammes of radium, and in eighty-four needles containing two, three or five milligrammes of radium element. The plates are screened with monel metal, the small tubes with 0·5 millimetre gold platinum alloy, and the last tubes with one millimetre gold platinum alloy. The screens of the needles consist of 0·5 millimetre platinum. The University of Sydney has now approved of the hire of radium to medical practitioners for the treatment of patients. This radium will be available for hire during the first week in August.

Diary for the Month.

AUG. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 5.—Victorian Branch, B.M.A.: Branch.
AUG. 6.—South Australian Branch, B.M.A.: Council.
AUG. 7.—Queensland Branch, B.M.A.: Branch.
AUG. 11.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 13.—New South Wales Branch B.M.A.: Clinical Meeting.
AUG. 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 26.—Victorian Branch, B.M.A.: Council.
AUG. 27.—South Australian Branch, B.M.A.: Branch.
AUG. 27.—New South Wales Branch, B.M.A.: Branch.
AUG. 28.—Queensland Branch, B.M.A.: Council.
SEPT. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 2.—Victorian Branch, B.M.A.: Branch.
SEPT. 3.—South Australian Branch, B.M.A.: Council.
SEPT. 4.—Queensland Branch, B.M.A.: Branch.
SEPT. 8.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 10.—New South Wales Branch, B.M.A.: Clinical Meeting.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Clinical Assistant.
COMMONWEALTH DEPARTMENT OF HEALTH: Medical Officer.
NATIONAL ASSOCIATION FOR THE PREVENTION AND CURE OF CONSUMPTION, SYDNEY, NEW SOUTH WALES: Honorary Physician.
SAINT VINCENT'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Assistant Dermatologist, Honorary Clinical Assistant to Dermatological Department.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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